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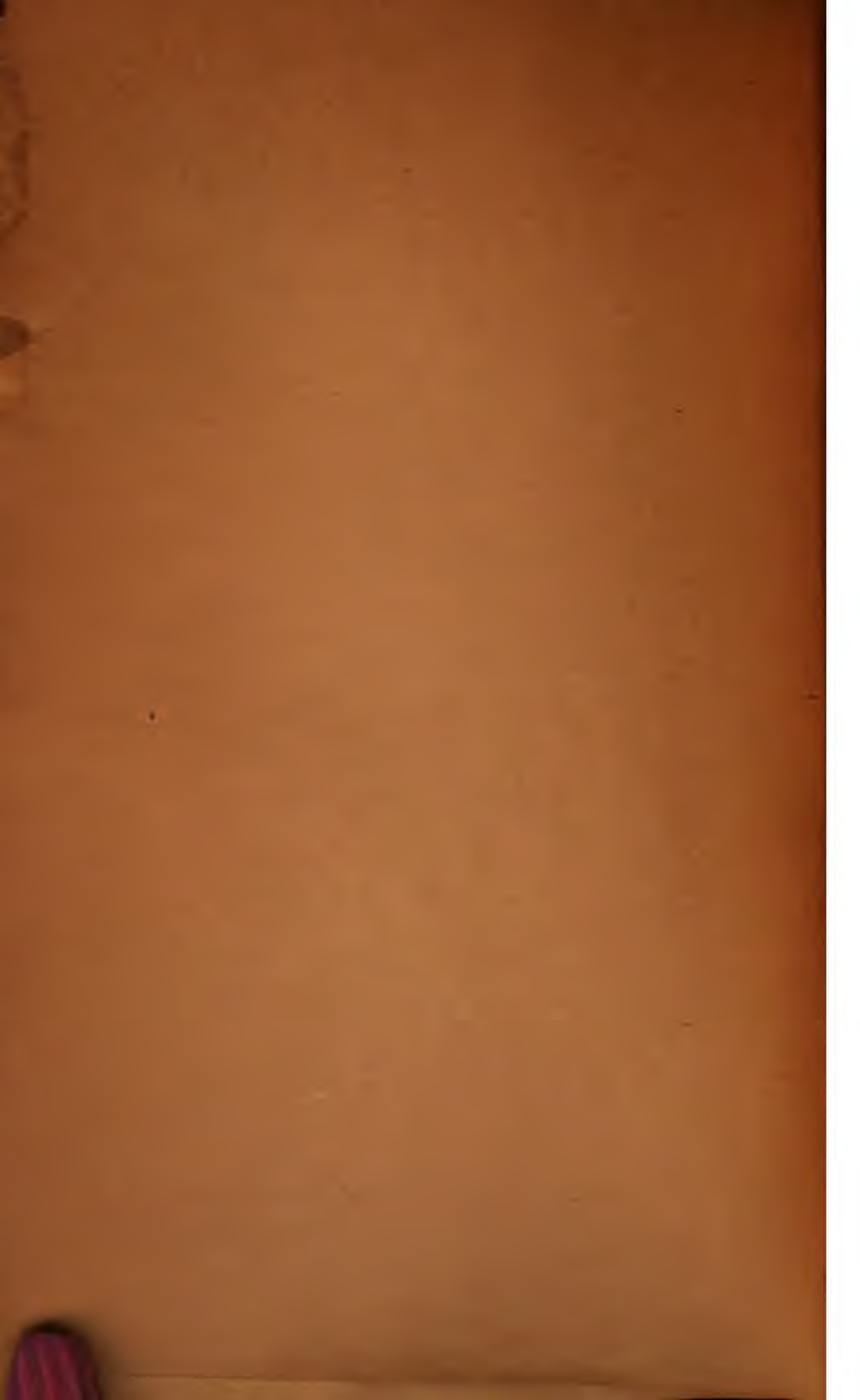
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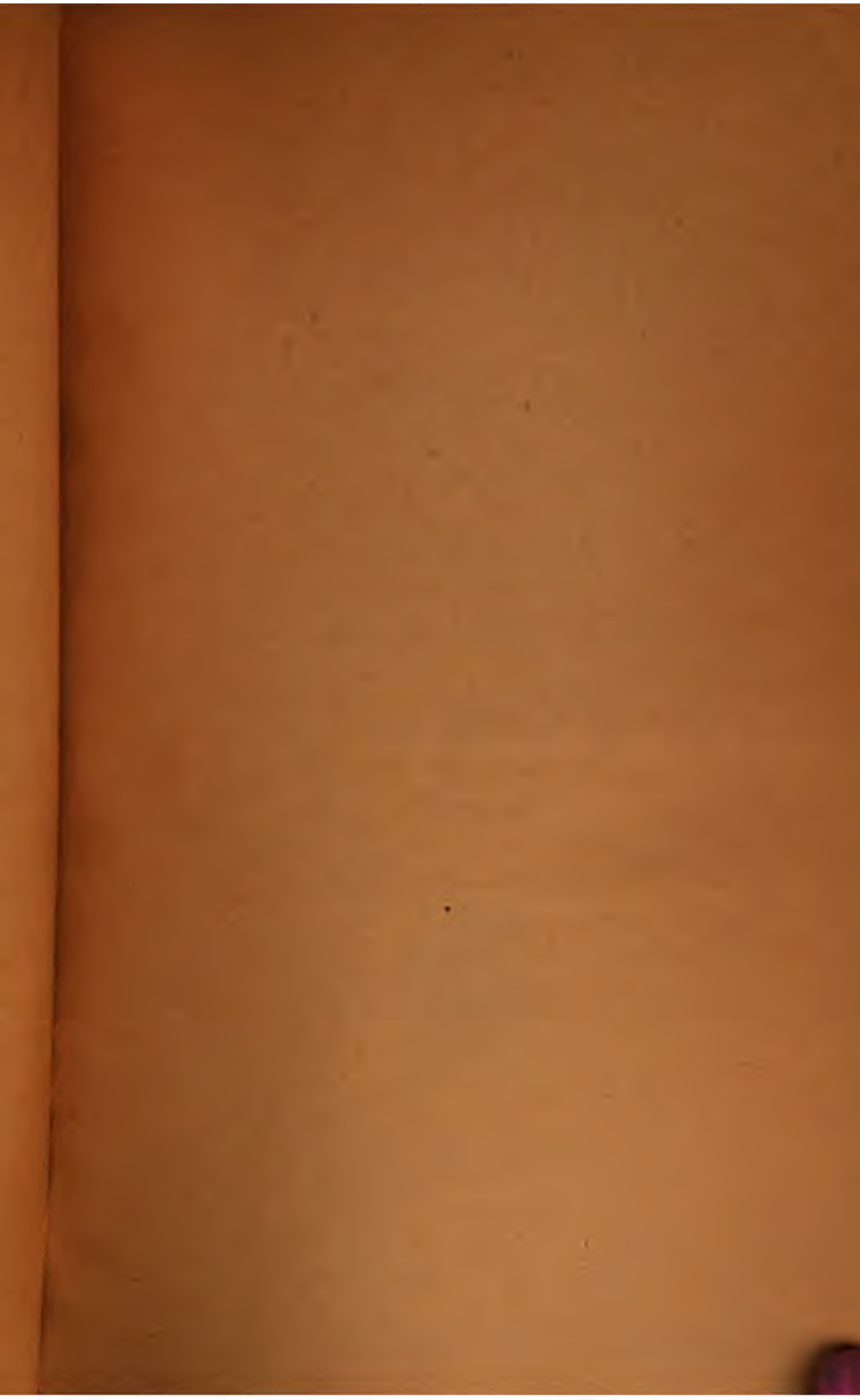
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VOL. XVI.—PART 1.

MARCH 1903.

THE
JOURNAL OF
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AND
THERAPEUTICS

(With which is incorporated The Veterinarian)

EDITED BY

J. M'FADYEAN, M.B., B.Sc., M.R.C.V.S.
ROYAL VETERINARY COLLEGE, LONDON

AND

JOHN A. W. DOLLAR, M.R.C.V.S.
LONDON



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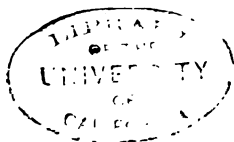
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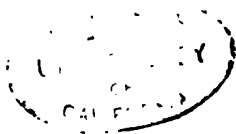
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THE TREATMENT OF COLIC IN THE HORSE BY
INTRAVENOUS INJECTIONS OF CHLORIDE OF
BARIUM.¹

By P. J. CADIOT, Veterinary College, Alfort, Paris.

THE use of chloride of barium in veterinary medicine was suggested to Dieckerhoff by the symptoms produced in two horses which had accidentally partaken of the salt. These animals, which worked in a railway station, were poisoned by licking the floor of a van on which some chloride of barium had fallen from the sacks containing it.

After studying the action on horses of chloride of barium administered through the medium of the digestive tract, the connective tissue, and the veins, Dieckerhoff finally chose the method of intravenous injection for treating colic, and in 1895 described this new treatment in an article in which he summarised the results of his first series of fifty-one observations. The investigations hitherto reported on the action of chloride of barium refer to its administration by the mouth in horses and the larger ruminants, and to its use in the treatment of different bovine disorders, such as dyspepsia, constipation, tympanites, and post-partum paralysis. The latter I leave on one side, intending at present to deal only with the intravenous use of this material in treating equine colic. Some months after the publication of Dieckerhoff's article his assistant, Brass, described a second series of 136 cases of colic treated by intravenous injections of chloride of barium. Of these, twelve horses died, that is, 9 per cent. The lesions found on *post-mortem* examination explained these fatal issues. Seven had twist of the large intestine, two volvulus of the small

¹ Translated from the "Recueil de Méd. Vét.," 15th October 1902.

intestine, one a hernia of the hiatus of Winslow, and one enteroperitonitis, the small intestine being strangulated by the epiploon. The doses injected into the jugular vein by Dieckerhoff and Brass varied between $7\frac{1}{2}$ and 19 grains. To heavy horses they several times gave doses of 22 to 30 grains. In 187 patients no fatal result referable to the treatment occurred.

When I introduced this new method of treating colic to the Central Society of Veterinary Medicine I had used it on thirty-two horses, injecting doses of the chloride varying between 5 and 18 grains into the jugular. Of these thirty-two patients, three died; two had rupture of the stomach, and one twist of the small intestine. No alarming symptoms occurred, there was no indication of toxic action, and the rapid effects produced strongly impressed me. Some minutes after injection the contents of the rectum were expelled, then followed solid, semi-liquid, and gaseous evacuations, repeated at more or less frequent intervals for one-half to one hour. The peristalsis and liquid secretions of the bowel were increased much more rapidly than by eserine, pilocarpine, or arecoline.

My own observations, like those made up to that period in the Berlin clinique, appeared to show that, despite its toxicity, chloride of barium could be introduced into the veins in the doses above indicated without danger, provided these doses were approximately proportioned to the size or weight of the animals; that is to say, for small animals the dose should vary between 5 and 10 grains, for those of medium size $7\frac{1}{2}$ and 14 grains, and for large animals between 12 and 18 grains.

None of the veterinary surgeons who used chloride of barium in the manner above suggested reported any fatal case or declared themselves disappointed with the results obtained. Dahlenburg treated thirty-two horses affected with colic by the intravenous injection of doses similar to those first suggested by Dieckerhoff. Having seen no serious symptoms attributable to the toxicity of the drug, he suggested that the deaths reported by others were due to errors in the solution or in estimating the dose. In forty-eight cases Grüner injected into the jugular doses of chloride varying between 12 and 15 grains without producing any alarming results. In two cases a dose of 30 grains and in another a dose of 45 grains only produced the usual effects, though in an intensified form. In the clinique of the Veterinary School of Buda-Pesth Hutyra during 1897 treated 191 horses by intravenous injections, the doses varying between 7 and 18 grains. The smaller dose was given whenever any sign of weak heart was observed in the subject. He reported no cases of rapid death, and no symptoms suggesting a toxic action in the medicine. Angerstein, Carrey, Lehnert, and Petersen have published less favourable reports.

But large quantities injected in one dose, particularly in concentrated solution, have produced fatal effects certainly due to the action of chloride of barium on the heart. Whatever Grüner, Plattner, Dahlenburg, and others may say, these results were due neither to impurities in the drug nor to changes in the solution, nor, it would seem, to errors in the method of administration. There is no doubt whatever that the majority were a result of this toxic character of chloride of barium and of its very special action on the

heart. Injected rapidly and in full doses into the blood, the chloride of barium reaches the left heart and cardiac arteries in an insufficiently diluted state, and by its direct action on the cardiac ganglia or on the muscular fibres may produce tetanisation of the heart and almost instantaneous death. In the majority of cases where death followed a few moments after injection the doses varied between 12 and 19 grains. Animals have died after even smaller quantities: Siebert's case was killed by 12 grains, Mollereau's by 9 grains, and Ries's by 7 grains. In France, the two fatalities mentioned by Mollereau appeared to suggest that chloride of barium is an extremely dangerous remedy which in practice should be avoided. But a study of the total number of cases hitherto published shows that in general its injurious properties have been greatly exaggerated, and that of the horses for whose sudden death it was undoubtedly responsible many were already on the point of dying or were very seriously affected by the absorption of toxic substances from the intestine.

Close observation shows that fatal results most often follow when the animals are in an advanced stage of the disease, when they show marked circulatory disturbance, the pulse and heart being very rapid and feeble, or the violence of the heart being in marked contrast with the feebleness of the pulse, or, again, when the patient is suffering from some old-standing cardiac mischief. In such cases, therefore, it is wise to administer only small doses, or to divide the dose into several parts.

Experience has shown that the injection of small doses, repeated in accordance with the progress of the case, produce all the good effects to be obtained from the drug. Since 1896 I have practised the following method: A first injection of $3\frac{1}{2}$ to 7 grains is given, followed, in a quarter of an hour, by another of 3 to 5 grains, which if necessary can be repeated fifteen or twenty minutes later. In the advanced cases of stoppage of the bowel, where the animal is already under the influence of intestinal poisons, it is wise not to exceed doses of 4 to 6 grains repeated three or four times at intervals of a quarter of an hour to twenty minutes.

Since 1896 I have treated, or caused to be treated, in the above way 162 cases of colic in the horse. In a certain number I have also bled the animals, applied stimulant applications or douches of cold water to the abdomen, and have given cold enemata. Of these animals twenty-one (13 per cent.) died, but I have never noted any toxic effects produced by the chloride of barium.

During the month of September last I systematically treated all horses sent here on account of colic by such injections. The solution used was of a strength of 1 in 30. Two or three injections were made at intervals of twenty to thirty minutes: in large animals a first injection of 12 cc. containing 6 grains of chloride was given, followed by one or two others of 9 to 10 cc. containing 15 grains; for horses of medium size, a first injection of 10 cc., followed by one or two others of 8 cc.; for small animals, a first injection of 8 cc., and one or two others of 6 cc. A review of the clinical records sent me by MM. Pécard and Rebeu, students in the clinique of pathological medicine, show that these small doses are sufficient to rapidly increase peristalsis and intestinal secretions.

Towards 5 P.M. the attack diminished, and the horse no longer attempted to lie down. It still turned its head towards the flank, but showed no other sign of pain. During the evening it was sent home cured.

Case VII.—Bay stallion, ten years old. Attacked with colic during the morning of the 26th September, and brought to the School at 10 A.M.

The patient showed signs of acute abdominal pain. The conjunctiva was slightly injected; the pulse was of normal volume but somewhat frequent. Examination of the inguinal region and of the posterior abdominal region per rectum revealed nothing abnormal.

At 10.10 A.M. 10 cc. of solution, containing 5 grains of chloride of barium, were injected, and the horse was walked about. Five minutes later it made efforts to defæcate, and ten minutes afterwards passed solid excreta. At 10.30 a second injection of 8 cc. was given. During the ensuing quarter of an hour there were three abundant liquid evacuations. Towards 11 A.M. the symptoms abated, and the animal no longer attempted to lie down. It was left at liberty in its box, and an hour later showed no sign of pain.

Case VIII.—Grey mare, twelve years old. At 3 P.M. on the 30th September this animal showed colic, and at 4 P.M. was sent to the School.

She attempted to roll. The conjunctiva was pale, the pulse full, the respiration rapid. The flank was slightly distended with gas.

At 4.30 P.M. 10 cc. of solution were injected into the jugular. In five minutes fæces and urine were passed, followed five minutes later by semi-liquid fæces.

At 4.50 P.M. a second injection of 8 cc. of the solution was made. Ten minutes later large quantities of liquid fæces were passed. The animal at once appeared relieved. It was sent home about 5.30 P.M.

Case IX.—Bay gelding, twelve years old. On the morning of the 1st October this animal received 7 litres of oats; it worked until mid-day, when it consumed 7 litres more, and, having slipped its head-collar, ate part of another horse's food. It was attacked with colic towards 2 P.M., and was sent to the College at 5 P.M.

It was greatly distended with gas, and continually looked towards its flank, lay down from time to time, and made ineffectual efforts to defæcate. The conjunctiva was pale, the pulse strong, the respiration slightly accelerated.

At 5.10 P.M. 10 cc. of the solution were given. In five minutes the animal twice passed fæces. At 5.30 a further injection of 8 cc. solution was given. Five minutes later there was an abundant passage of liquid excreta.

Case X.—Dappled grey stallion, eight years old, 16½ hands high. This animal returned home on the 1st October at 11 P.M., and ate its ordinary allowance of food. Next morning at 4 A.M. it had its breakfast. Towards 8 A.M. it showed colic. Despite free bleeding it became worse. At 9 A.M. it was brought to the School.

It was very restless, lay down, struggled violently, and occasionally eructated. The conjunctiva was injected, the pulse feeble, the respiration rapid. Examination of the genital organs showed nothing abnormal.

At 9.30 A.M. it received an injection of nine-tenths of a grain of nitrate of pilocarpine, followed, ten minutes later, by an injection of four-tenths of a grain of sulphate of eserine and by repeated enemata of soap and water containing sulphate of soda. Salivation was abundant, but the bowel failed to act and pain remained acute. At 10 A.M. a mustard plaster was applied to the abdomen and oil of turpentine was rubbed into the skin over the lumbar region. The animal struggled so violently that it was difficult to approach it.

At 11 A.M. 10 cc. of the solution were injected into the jugular. In five minutes two evacuations occurred. At 11.20, 8 cc. of the solution were injected. The animal soon afterwards passed gas and frequent borborygmus was heard. In ten minutes it passed semi-liquid faeces and five minutes later liquid material. Finally the pain diminished, and the other symptoms gradually disappeared.

Given in divided doses, chloride of barium appears very active, prompt in its effects, and without danger. At the Berlin School Brass and Witt have given more than 30,000 injections without a single fatal result and without symptoms of poisoning. No accidents occurred at the Claye Farm, where M. Cluzet treated 445 cases of colic among the horses of the Paris Omnibus Co. with doses of 4 grains repeated two or three times at intervals of a quarter of an hour to half an hour; and almost all practitioners who have continued to employ this drug with the precautions just suggested report having had excellent or at least very satisfactory results.

One remark may be added concerning the method of injection. In addition to injecting the solution slowly, it is important to avoid introducing air into the vein. Although the passage of two or three bubbles is not of consequence, any considerable quantity might lead to death. Such a result would certainly be exceptional, but its possibility must not be lost sight of, even although experience has shown that the injection of considerable quantities of air does not commonly produce any accident. After describing the results of his injections of chloride of barium, Grüner adds that he has introduced six syringes full of air into the horse's jugular without observing any peculiar symptoms whatever. This experiment and those just alluded to are only in the nature of negative facts. Recorded cases clearly show that the entry of a certain quantity of air into the horse's jugular may be very dangerous, if not fatal.

EXPERIMENTAL RESEARCHES REGARDING BLACK-QUARTER.¹

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IN a previous article we have described some researches regarding immunisation against black-quarter by means of pure culture, protective serum, and the combined employment of cultures and serum. We shall here describe, together with some new experiments, the results obtained by employing these different methods of vaccination in practice.

¹ Translated from the "Annales de l'Institut Pasteur," December 1902.

I.—Vaccination by Means of a Single Inoculation of a very Attenuated Pure Virus.

An experimental study of the methods of conferring immunity against black-quarter led us to formulate the following conclusion:—

“The employment of pure virus will without doubt enable one to vaccinate by a single operation, and this is a method which apparently ought to be preferred on account of its safety and simplicity.”

As a matter of fact, the method responded exactly to the requirements of practitioners, who wished to have only a single operation, and to suppress all manipulation in the preparation of the vaccines.

The employment of pure products appeared to furnish a guarantee of the safety of the operation. The practical tests which were carried out on cattle kept in the premises attached to the laboratory showed that they hardly reacted at all when they were inoculated under the skin of the shoulder with from 2 to 3 cc. of culture that had been heated at 70°, 65°, or even at 60° C. When tested nine days afterwards by the intramuscular injection of virulent juice, which killed the control subject in thirty hours, the vaccinated animals were found to be completely refractory.

It seemed as if the operation might be practised with perfect safety in all conditions, and thirty-nine animals were tentatively inoculated in this way. The immediate results were bad. Four of the vaccinated animals died from black-quarter, attributable to the inoculation. The survivors were solidly immunised, since none of them contracted the disease, although kept amid surroundings that exposed them to serious risk of infection.

The details of this experiment have already been published in our article on the accidents following vaccination. They showed that a method of vaccination which might be quite safe for animals free from any anterior contamination may be dangerous for subjects exposed to infection in contaminated districts.

A method of immunisation that would be at once effective, lasting, and free from danger, remained to be discovered. Two new solutions of the problem offered themselves:—

(1) To abandon the attempt to confer immunity by a single operation, and to operate on two occasions with pure liquid vaccines of different strength, the first vaccine being as attenuated as possible.

(2) To combine the injection of protective serum with the inoculation of vaccine, according to one of the methods previously used by one of us in the case of swine erysipelas.

II.—Two Operations with Pure Liquid Vaccines.

Owing to the extreme susceptibility of bovine animals in the state of latent infection to virulent experimental inoculation, one is obliged to employ a feeble virus for the first vaccination. On the other hand, it is necessary to produce a vaccinating effect. A vaccine that is too feeble will leave the system unprotected, and the second vaccine will then provoke accidents. We have previously shown that cultures heated to 80° do not give rise to any accident in a guinea-pig, and that they do not confer immunity. The following experiment proves that that is true also for the ox.

A Breton cow received under the skin of the neck 10 cc. of a deposit from a rich sporulating culture which had been heated for two hours on 80°. The material inoculated contained many millions of spores. No trace of reaction was observable, and when the animal was tested fifteen days afterwards by a virulent inoculation it died from black-quarter in forty-eight hours.

We use as a first vaccine a pure culture heated for three hours at 75°, this being capable of exciting a very slight reaction in the inoculated animals. The second vaccine is obtained by an exposure to 68°-70° for the same time.

The dose of each vaccine for adult animals is 1 cc., and half that quantity for animals under a year old. The inoculations are made with an interval of eight or ten days, under the skin behind the shoulder, at the flank, or at the junction of the middle or upper third of the tail.

1002 animals in different districts have been vaccinated in this way: Seine-Inferieure, 568; Nièvre, 182; Cantal, 133; Hautes-Pyrenees, 89; Nord, 30. All the vaccinated animals belonged to herds which annually lost between 10 and 20 per cent. of their total number.

In some cases the vaccination was carried out after a fatal case of quarter-evil had occurred either in the immediate neighbourhood or in the same herd. The animals vaccinated were thus exposed to risk of infection, the majority of them being contaminated.

All the vaccinated animals have escaped black-quarter, while the non-vaccinated animals around them have died from that disease. Moreover, the results obtained with the single vaccination showed that the pure cultures conferred solid immunity.

The chief interest of the experiment lies in the immediate results of the operation. Seven animals died, viz., one after the first vaccination, and six after the second. In some of these the lesions were generalised. All these accidents occurred among the subjects inoculated at the tail. Those inoculated at the shoulder did not exhibit any disturbance.

This series of experiments proves that pure vaccines, even when they are very attenuated, are not free from all risk. The results are not better than those obtained by the impure powdered vaccines, and this fact proves indirectly that the accidental impurities play only an insignificant rôle in the production of the accidents following vaccination. In almost every case these accidents are attributable to infection by the germs which are always present in animals kept in infected places.

III.—Vaccination by the Combined Employment of Serum and Virus.

(A) *Vaccination by Mixtures of Serum and Virus.*—Several years ago it was shown by one of us that a mixture of serum immunising against swine erysipelas with a virulent culture of the bacillus of that disease when inoculated under the skin or into the veins did not kill susceptible animals, and without danger conferred on them a firm and lasting immunity. It was subsequently shown that a mixture containing the malignant oedema bacillus and immunising serum was not fatal to the guinea-pig, although, contrary to what happened in the

case of swine erysipelas, no lasting immunity against malignant oedema was thus conferred.

On the other hand, we have proved that a mixture of black-quarter virus and serum does not kill the guinea-pig and does not immunise it. Arloing made a similar observation with regard to sheep, but he thought this same mixture conferred immunity on the ox.

A little later Arloing recommended a method of vaccinating cattle based on the employment of two powerful vaccines of different strength, inoculated at the same time, after mixing them with one-tenth cc. of protective serum.

In his note to the Académie des Sciences Arloing described only his experiments on sheep. The addition of serum to a very small quantity of attenuated virus would be capable of "moderating the immediate effects of the vaccine while not interfering with its immunising effects."

What we know with regard to the susceptibility of animals kept amid infected surroundings to even the most feeble vaccines enables one to foresee the danger of employing simultaneously strong vaccines, even when they are combined with a homœopathic dose of serum. Moreover, one cannot compare the mixture of powder and serum prepared at the moment of its employment with a mixture of serum and culture. The small quantity of serum inoculated becomes absorbed before the spores are able to germinate, and even before phagocytosis has been called into play. The operation is thus similar to the inoculation of strong powdered vaccines into an animal treated with one-tenth or one-fifth cc. serum, which is manifestly an insufficient dose, nor can one admit that the sensibiliser of the serum can be fixed by the spores in a few minutes when these are enclosed in scales of cooked albumen. On *a priori* grounds the method thus does not appear to be without danger, and it is doubtful if one dare take the risk of putting it into practice. Experience has just shown us that it is uncertain in its results.

The experiments made by Arloing at Nevers in September 1900 were themselves quite convincing with regard to this point, since some of the vaccinated animals succumbed to the test inoculation. We shall here give some new experiments on the point. In these we used a very strong serum obtained from the horse.

Cow XX.—Injected under the skin of the shoulder a mixture of 10 cc. of serum and 10 drops of virulent serosity. Died from black-quarter after fifty hours, with scarcely any local lesion, but almost all the muscles invaded.

Cow XXI.—Injected under the skin of the shoulder a mixture of 2 cc. of serum with 10 drops of virulent serosity. No injurious effects followed.

Guinea-pig 400.—Injected into the thigh 5 cc. of serum mixed with 5 drops of virulent serosity. Died in thirty hours.

Guinea-pig 401.—Inoculated at the same time as the preceding with 2 cc. of serum and 5 drops of serosity. Resisted.

Cow XXV.—Injected under the shoulder a mixture of 2 cc. of serum with 10 drops of virulent serosity. Died of black-quarter in seventy-two hours, with no local lesion, but all the muscles altered.

Cow XXVI.—Injected under the skin of the shoulder a mixture of 2 cc. of serum with 6 drops of virulent serosity. Resisted.

The mixtures of serum and virus are thus inconstant in their immediate effects in the ox. Moreover, the same variability is

observed in the guinea-pig, as we have already pointed out. On the other hand, the subjects which resist this treatment are not with certainty immunised. The proof of that is furnished by the following experiments.

Cow XXI (see above).—Had resisted inoculation with a mixture of 2 cc. serum and 10 drops of serosity.

Cow XXVI (see above).—Had resisted inoculation with a mixture of 2 cc. serum and 6 drops of serosity.

Eighteen days after the vaccination the two cows were tested with the same dose of virus.

Cow XXI.—Succumbed.

Cow XXVI.—Resisted, although when vaccinated it had received a smaller dose of virus.

It is thus demonstrated that inoculation with a mixture of serum and virus is equally uncertain in its immediate effects and in its remote results. The substitution of powdered impure virus for the pure liquid virus employed by us can only increase the uncertainty and inconveniences of the operation. This method of vaccination, being both dangerous and uncertain, deserves to be entirely rejected.

(B) *Vaccination by Successive Inoculation of Serum and Virus*.—Kitt's first experiments showed that the sheep acquires a solid and durable immunity when it is successively inoculated with serum and virus. Arloing confirmed this view, and showed that the same result is obtainable in the ox.

A priori one would think that the method of successive inoculations of serum and virus ought to avoid the risk of immediate accident from the vaccines, these being very attenuated. One of us has shown the benefit which may be derived by employing such a method of vaccinating against swine erysipelas.

In a previous article we have indicated the ordinary way in which accidents following vaccination arise in infected districts, and the rôle played by "antecedent latent infection" in their production. The previous injection of serum ought to have the effect of freeing the system from the germs which it contains, or at least of exalting for the moment its resistance, and thus of assuring the harmlessness of the subsequent inoculation with vaccine.

As a rule, when an ox receives under its skin 10 cc. of serum, and then twenty-four hours afterwards 10 drops of virulent serosity, it presents an intense thermal reaction (more than 2°), which lasts for two or three days. The local reaction is manifested by a flat œdematous swelling as large as the hand. There is no general disturbance, and the appetite is retained.

The animals which react in this way acquire a solid and durable immunity. Subsequently they resist inoculation with 1 cc. of virulent serosity into the muscles. It would certainly be dangerous to employ ordinary virus for the vaccination. Some experiments already made show that the results of such inoculation might be disastrous in practice.

Our experiments on vaccination have been carried out in the following fashion. The animals (young cattle) receive an injection of from 10 to 20 cc. of serum according to their weight.

Then five to eight days afterwards they are inoculated sub-

cutaneously at the shoulder, flank, or tail, with 1 cc. of pure culture which has been heated for three hours at 70°. We have employed two sera, one being double the strength of the other.

In different districts a total of 648 animals have been vaccinated : Seine-inferieure, 87 ; Nièvre, 373 ; Tarn-et-Garonne, 88 ; Nord, 75 ; Cantal, 25.

The results of the operation are indicated below :—

<i>Animals treated.</i>	<i>Losses after the vaccination.</i>	<i>Losses during the year.</i>	<i>Losses before the vaccination.</i>
Weak serum, 201.	8, or 4 %.	0.	10 to 20 %.
Strong serum, 447.	0.	1, or 22 %.	10 to 20 %.

It will be seen from this that it is necessary to employ rather strong serum in order to avoid immediate accidents. When this condition is complied with one has nothing to fear in any circumstances.

It ought to be observed that this method permits one to intervene successfully and without danger in places that are seriously affected, that is to say, in conditions where vaccination properly so-called is attended with risk of accidents and assures only a tardy immunity. Inoculation with serum immediately protects the animals from all risk of infection. The vaccination by our method has always been resorted to in herds which had just lost one or several animals, and the mortality has always ceased immediately after the injection of the serum.

The strength of the immunity conferred by a single inoculation of a strong vaccine is demonstrated by the above statistics. Out of a total of 648 vaccinated animals there was only one death during the year following the operation, that is to say, the insignificant percentage of .15 per cent., and this although the herds were kept in seriously infected districts. In one instance, fourteen animals were vaccinated while two on the same premises were unvaccinated. These two died from black-quarter, while the fourteen others remained healthy. Should a more extended experience show that the immunity conferred is insufficient or too short in certain places, it will be very easy to reinforce it by carrying out, ten or twelve days after the injection of the first vaccine, a second inoculation with a virus that has been heated only to 65°.

The object of our researches was to discover a method of vaccination that was at once simple and certain, and we have arrived at the conclusion that these two qualities are incompatible. In order to safely confer immunity at least two operations are indispensable, and these require the preparation of an immunising serum and two pure attenuated viruses.

It is easy to foresee the circumstances in which the simple methods and the certain methods will be preferable.

In countries where the cattle are not of any great value it is possible to sacrifice everything for the sake of simplicity in the operation. The rapid methods will also be applicable where the animals are resistant to the virus. Hence it is that these methods have been welcomed in the United States, in Algeria, and in some parts of Italy. In these conditions one may inoculate at the tail with almost any virus, and the empirical methods of operating triumph over all others. In some other countries and in France the condi-

tions are quite different. The animals usually kept have almost always a high value, and owing to the sub-division of property any losses sustained by small owners fall very heavily on them. In these circumstances, the main thing is to have an operation free from danger. Vaccination is abandoned for a long time after it produces an accident.

The method of vaccination which we have described above permits one to vaccinate cattle with certainty and safety against black-quarter. We believe it would be useless to recount here the advantages which it possesses over the methods hitherto employed.

To sum up:—

A single inoculation with a more or less attenuated pure vaccine, although realisable experimentally, is in practice attended with the risk of serious accidents.

Vaccination by means of two operations with pure vaccines, even when these are very attenuated, is not absolutely safe.

Vaccination with a mixture of immunising serum and virus is attended with the risk of immediate accidents, and it does not immunise with certainty.

The method which ought to be preferred consists in successively inoculating with immunising serum and a pure attenuated virus.

EPIZOOTIC ABORTION IN COWS AND ITS PREVENTION.

By J. PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

TECHNICALLY speaking, it is customary to make some distinction between miscarriage, abortion, and premature birth, but for all practical purposes it may be taken that "abortion," as it affects our subject, means expulsion of the fœtus before its time and usually before it is capable of living after birth. It is an occurrence to which all mammals are liable, but experience tells us that among the domesticated animals it is much more frequent and serious as affecting cows and heifers. Conditions connected with the occurrence are so varied that abortions have come to be classified under two heads, "Sporadic" or "Accidental," and "Enzoötic," "Epizoötic," "Plague-like," or "Contagious."

Pregnant females of all species may, in isolated instances, cast their young as the result of some accident, such as a mechanical injury, some mental impression, etc. Sometimes such a cause affects but a single animal, while at other times it may affect a considerable number, *e.g.*, narrow doorways, tympanites, fright, some poisons or infectious diseases, as Cattle Plague, Foot-and-Mouth Disease, or Pleuro-pneumonia. There is often an appreciable connection of cause and effect, and evidence that one abortion is not dependent on another abortion, though many be induced by a common cause. Such abortions cease when the evident cause ceases to exist. They are all placed in the category "Sporadic" or "Accidental." Under the head "Epizoötic," "Enzoötic," "Plague-like," "Contagious" are classed those abortions which usually affect many animals in a herd, flock, or stud, whose existence usually appears to be more

or less dependent on some former case, and until recently the relation of cause and effect has been obscure. The terms "Picking," "Slinking," and "Slipping" or "Warping" are most commonly applied to the latter form.

This malady is recognised in the most ancient writings on the subject, and though it is very commonly believed that the epizootic form is now more general and on the increase, there is ample evidence to show that in very early times its occurrence in flocks and herds was a matter of serious consideration. In Genesis xxxi., v. 38, Jacob says to Laban, "All the twenty years that I have been with thee neither the ewes nor the she goats have cast their young." Mascall, our oldest English writer on stock, in his book on *Cattle*, published in 1567, gives directions "how to keep cows which are great bellied in calf."

For the two subsequent centuries the records of cattle disease are very scant, but Lawrence, writing in 1805, says "Cows are well known to be much given to abortion, slinking or slipping their calves in an early period of gestation; it is sometimes epidemic, and thence some people have supposed it to be contagious."

Three years later, Skellett, referring to abortion, remarks "it is an accident to which cows are very liable, it has often been known to spread like an infectious disease and great losses have been suffered by cow-keepers from the same." In 1837 Jonati tells us that abortion spread through dairies and flocks, and mares were often affected; and a little later writes fully on the occurrence of enzoötic abortion in cows, ewes, and mares. In 1851 a prize essay written for the Royal Agricultural Society by Barlow states "abortion of cows is of such extensive prevalence amongst large stocks of cattle as fairly to be considered an epizootic disease." In 1860 we learn from Musgrove that "the district of Hereford abounds with abortions both in bovine and equine species." In 1869 and 1870 we have records of "serious epidemics amongst cows, mares, and ewes in Scotland."

Abortion among cows had in 1886 become so prevalent and serious that a question was asked in the House of Commons as to the desirability of bringing it under the Contagious Diseases (Animals) Act. In 1894 the Royal Agricultural Society appointed a Committee to enquire into circumstances connected with its common occurrence, and urged the Minister of Agriculture to take steps to check it.

It is to the so-called "Epizootic Abortion" that our attention is directed. The loss and disappointment associated with this affection are often extreme, and difficulties encountered in attempting to arrest its progress have led in many instances to the abandonment of breeding. As was before remarked, there are no statistics on which we may rely as to the extent of its prevalence or to form an opinion as to whether it is more common to-day than heretofore. We, however, know that its effect is sometimes such that for one or two years not a single calf in a herd is carried to full time. It has been asserted that of the cows in some districts as many as 60 per cent. abort. It is, however, not often that over 50 per cent. of a herd abort. Undoubtedly some modern conditions favour its spread, but the facts which have been adduced from history will, it is to be hoped, be somewhat reassuring, for if a century since the disease had assumed such alarming proportions as indicated by the contemporary writers,

when no special means have been adopted to check its extension, unless there were in operation some natural hindrance the breeding of cattle must have ceased long ago.

It is not, however, its existence, but its prevention, which most concerns us, and in order to be in the most favourable position to adopt and carry out preventive measures it is well to appreciate what is known as to its nature and cause. It is when the cause and effect are distinctly associated that we are able and disposed to energetically direct our efforts to the definite object of attacking the cause.

The contents of the womb may be expelled as a result of impressions on the nervous system through some of the ordinary or special senses, inducing the state of fright, etc., bringing about violent contractions of the womb or "heaving." Expulsion, however, usually results from some changes in those contents or of the relation between them and the womb. Under natural conditions, as soon as the foetus is capable of a separate existence it becomes independent—it is not a part of the being of its mother, but a foreign body and an irritant unfit to remain there and is cast off, in accordance with a principle exemplified in coughing, vomiting, sneezing, etc. Anything, therefore, which interferences with the vitality of the foetus, or breaks down the relations of the foetus to the womb, will bring about expulsion of the former.

Without regarding abortion in this light, the greatest variety of circumstances have been brought forward to account for its occurrence. Witchcraft, sympathy, smells, sights, frights, domestication, sewage, diet, climate, even evil spirits and a thousand other agencies, have been accredited with the power. The theory of contagion, however, had a century ago forced itself on many observers, the majority of whom were quite unwilling to accept it, and while advocating the application of disinfectant measures, took the trouble to state that it was not on the ground that affection was due to contagion, but to sympathy. In more recent times, with improved methods of observation and increased scientific knowledge, the view of a contagious nature had become firmly held, and epizootic abortion, like some other diseases, as tuberculosis, was generally recognised as contagious long before the causal germ was identified.

The publication (in 1896) of the results of experiments made by a celebrated Danish veterinary surgeon, Dr Bang, and his discovery of the germ, placed the matter on a more satisfactory basis. This observer discovered in the envelopes of the foetus and between them and the walls of the womb during life, before abortion took place, also in the abortion, afterbirth, and discharge from the genital passages of the animal which had aborted, certain bacilli or germs. By introducing these into the genital passages or into the veins of healthy pregnant cows abortion was induced. Though conclusive evidence has not yet been forthcoming, it is thought possible that the same effect may be induced by swallowing the germs with food or water, or even by their entrance into the air passages. It is, however, highly probable that under natural circumstances the germs enter by the vagina. There is every reason for supposing that a large proportion of cows to whose womb the germs gain access abort, but it is possible that in rare instances a cow's womb may be infected and contain the germs and

yet abortion not follow, so that a cow which has not aborted may possibly be dangerous as a means of spreading the disease.

Epizootic abortion of cows is therefore due to a disease of the womb and membranes of the foetus induced by germs, at any rate usually discharged by an infected cow. This reservation is made because though there is no room for doubting that in the great majority of cases the germ is derived from an affected cow, we are not yet in a position to say positively that such germs may not be derived from other sources, though, it must be said that our experience does not support such a view. It cannot, however, be too emphatically stated that the infected cow is the paramount danger, and that our attempts to prevent must be based on an appreciation of this fact. She remains a source of mischief as long as she discharges or harbours the virus. There is also good reason for believing that the germs retain their power of inducing the diseases some time after they are discharged.

It is only by preventing access of these germs to healthy animals that we may hope to prevent occurrence of the disease.

Anything contaminated with discharge from an infected cow may prove a medium for the conveyance of the disease to the healthy cow. It will then be easily understood how the floor, etc., of a byre, or a spot of pasture where abortion has taken place, or where an infected animal stands, attendants, certain parts of the cow, especially the tail, or a bull having served an infected cow, etc., may be a source of danger.

It is not yet proved whether non-pregnant animals may contract the disease in their wombs and so become, though unsuspected, means of propagating the malady; but, as there is apparent no specific reason why they should not, a certain amount of suspicion must rest on even non-pregnant animals which have been exposed to the chances of infection.

The period which elapses between the access of the germ to the healthy animal and the occurrence of abortion is a matter for further inquiry. From the reports of experiments already carried out, this period would appear to vary from twenty-one days to ten weeks, *i.e.*, when the material has been introduced into the genital passage. It must not, however, be concluded from the result of experiments, in which the infecting matter has been directly and purposely inserted into the genital passages, that the germs will always be acquired so readily when healthy animals are merely brought into contact with the affected, that is to say, by simple cohabitation.

It is equally important to bear in mind that a short period of cohabitation with aborting cows *may* be sufficient to infect those previously healthy. Naturally this period must vary widely, so that after the introduction of an infected animal into a healthy herd it is not possible to know when other cows will abort. It has been known to occur six weeks after such introduction.

Even from experimental cases it has been observed that the effect of introducing the germs into pregnant cows is not always the same; indeed, in some instances there is no appreciable effect, and the calf is carried to full time. Under natural conditions we observe the same variation after exposure to the chances of infection, and it must be remarked, as one of the most noticeable and important characteristics

of the malady, that in the course of an outbreak a certain degree of immunity is acquired. It would seem to be similar to the course observed in small-pox and some other well known contagious diseases, in which one attack confers some protection. Indeed, it is one of the most satisfactory points to note in connection with this scourge that there is a natural tendency for it to exhaust itself and die out in the course of from three to five years. This natural beneficial course is, however, interfered with if any fresh animals are introduced into the aborting herd. If such a practice is persisted in the outbreak may continue indefinitely. The explanation of this circumstance is not clear. We do not know how long acquired immunity lasts; it may be that cows again become susceptible, or perhaps the germs acquire fresh power by passing through freshly introduced cows.

The effect of the disease on the system of the cow is often trifling; indeed, it frequently happens that the first evidence of a cow's having aborted is the on-coming of *œstrum*, which usually occurs from three to eight days after. This, of course, is usually in the earlier cases in an outbreak, and unfortunately it permits of contamination of the herd before the existence of the disease is suspected.

When it is recognised that the disease is established in a herd, and a sharp look out is kept, occasionally some time before the expulsion of the womb contents there may be noticed some change in the quantity and quality of the milk, or a brownish discharge from the genital organs. Rarely is there any marked constitutional disturbance to indicate its approach.

The measures which have been at different times adopted to prevent or arrest the affection have been on a par with the suggested causes. Among the more common it may be of interest to recall the horse shoe, the killed fox, or magpie nailed to the cow-house door; the goat kept in the byre, and the burial of an abortion at its threshold. Abortions were believed to be due to some inscrutable mystic agencies, and equally unintelligible measures were resorted to to counteract their influence.

Preventive measures must, however, be founded on the knowledge that the disease is contagious, and must be carried out under the conviction that our herds can only be maintained free from the risks by preventing access of the germs—the essence of this contagious quality. When considering measures for preventing the introduction of the disease into healthy herds, the fact will be borne in mind that the cow or heifer which has aborted, or is already affected with the malady of which abortion is the result, but still pregnant, and the bull which has served aborted cows, must be a ready means by which the germs may be transmitted from one situation to another, and that it is not yet proved that healthy animals which have been associated with the affected may not carry the contagion.

The matter is often surrounded with insurmountable obstacles, and inasmuch as there are no means of identifying dangerous animals it is impossible to avoid all the risks of contaminating a herd except by not introducing fresh animals or by purchasing only from herds free from abortion. Under present conditions this is rarely practicable, for dairies and breeding herds must be replenished, and to trace the origin of animals bought in open market before purchase is not often possible. It is to be feared that it is commonly regarded the easiest

way out of the trouble to sell cows which have aborted, and this is, I believe, a common practice. It is hard to conceive of any means by which greater injury can be done to a breeder than by the introduction of the germ of this affection.

There is now no question as to epizootic abortion being contagious and capable of being conveyed by animals from aborting herds. The losses to stock-breeders and the country at large are serious and apparently on the increase. The time has arrived when it should be legally regarded as a criminal offence to sell an animal from an aborting stock without stating the fact to the intending buyer. I think the Minister of Agriculture would be justified in using means for bringing the affection within the operation of the Contagious Diseases (Animals) Act. This would supply a partial if not complete safeguard against the introduction of the malady into healthy situations, an event now not under the control of the breeder. Isolation of new animals is not often practicable, and no cow from an aborting stock may be said to be safe. It is, however, advisable that newly purchased animals should be kept as far as possible from the chance of affecting pregnant cows. Thus, they may be placed in the lower portion of a byre, so that should abortion take place or contaminated matter be discharged from the genital passages they would be less likely to reach the healthy. Barren cows should always be suspected.

Next to keeping the malady out of a herd comes the consideration of eradicating it, or arresting its progress, when established, and this can only be effected by preventing its spread from the affected to the healthy, for to attempt to avert the expulsion of the fœtus after the womb is infected is useless, if not impossible. The difficulties encountered in such an attempt are by no means light. We have no means of ascertaining when a cow is infected and so to a greater or lesser degree dangerous, so that it must sometimes happen that before any animal is at all suspected she has spread the disease to her fellows. Inasmuch as it cannot be determined except by bacteriological examination if any abortion be due to contagion or other cause, it is well to treat all as though contagious.

It is at the period of abortion and that immediately following it that the aborting animal is most dangerous. The abortion, afterbirth, and discharges from the genital passages swarm with the germs. The cow should be isolated on manifestation of the earliest symptoms of approaching abortion, or of having aborted. The fœtus and its membranes should be destroyed by fire or chemicals, or buried out of reach of cattle. The place in which abortion occurred should be thoroughly disinfected; and, while it cannot be doubted that the more thorough the disinfection the greater are the chances of success, the gutter, the floor, etc., under and behind the cow, should receive special attention.

As carbolic acid and some other disinfectants have strong odours and are liable to taint the milk, it is advisable to use a solution of sulphate of copper for this purpose. All cows in the shed should have their external genitals, tails, and back parts generally, sprayed daily with a disinfectant, such as solution of lysol or chloride of zinc, with a view of destroying any germs possibly resting there. Powdered lime freely used on the floor is believed to be very useful. Manure from byres where abortion has taken place should not be used for pastures

on which cows will graze. Should abortion take place on the pasture it should be similarly dealt with, the cow being brought into the house and kept isolated. All cows which have aborted should have the vagina syringed out daily with some disinfectant solution as 1 per thousand mercuric chloride or 2 per cent. of carbolic acid. It is also advocated that the womb itself, the source of the germs, should be washed out three times a week for three weeks after aborting, but this should not be undertaken except under direction of a veterinary surgeon, as alarming straining sometimes follows the injection, though no serious results have come to our knowledge.

It must not be forgotten that attendants may convey the contagium, and that isolation implies the attendance of some one who does not come in contact with non-aborting animals.

Bulls known to have served cows which have aborted should not be used for others. Their parts should be syringed and sponged with some non-irritating disinfectant daily, or at least after each service, and it will not be safe to use the animal for service of healthy cows for some weeks.

The next consideration is, what to do with cows which have aborted. They usually come into œstrus a few days after aborting, and, if then served, almost invariably abort again. Service should not be attempted for at least three months, during which time disinfection should be carried out. Once having aborted, there is usually a tendency to do so again if service occurs soon after, but this tendency diminishes as time goes on, and a certain degree of immunity follows or the germs become less virulent. The method of dealing with such cows will largely depend on the numbers to be dealt with. If only a small number, fattening for the butcher, while being strictly separated from the healthy, is probably the most radical and economical measure.

New purchases should be most carefully kept from chances of contamination, for though there appears a natural tendency for the disease to die out after from two to five years, the introduction of fresh animals appears in some way to maintain its existence.

The adoption of the foregoing measures in their entirety involves an immense amount of trouble and annoyance. In some instances it may not be possible; while success will largely depend on the thoroughness with which they are carried out, it is advisable to apply treatment on the same lines as far as circumstances may permit.

ACCIDENTS CAUSED BY "SURFACE-CONTACT" ELECTRICAL TRAMWAYS.¹

By P. J. CADIOT, Veterinary College, Alfort, Paris.

THE first "surface-contact" electrical tramways laid down in Paris and its environs passed through streets in which electrical energy was distributed by two systems, known by the names of their inventors, the system Diatto and the system Claret-Vuillemier. The latter has not become popular. At the present time almost all the surface contact lines are equipped with the Diatto system.

¹ Translated from the "Recueil de Méd. Vét.," 15th May 1909.

Considered from the points of view of installation and action, these two systems, despite an apparent analogy, are in reality very different. In both an underground cable extending throughout the system conveys the electric current from the generating station ; this cable communicates with metallic plates (surface-contacts, *Transl.*) in the roadway from which the energy is transmitted to the moving vehicle at the moment when the latter passes over the contact, and the surface-contacts are placed at such distances as to allow the current to be continuously supplied to the motors of the vehicle. At this point the resemblance between the two ceases. In the Claret-Vuillemier system automatic distributors are introduced between the cables and the surface-contacts, and each of these automatic distributors is connected to a series of thirty or forty surface-contacts, to each of which in succession it sends a current at the moment when the vehicle is passing over it. In the Diatto system each surface-contact receives the current from the supply cable directly, and is provided with an automatic accumulator apparatus which conducts the current to the metallic cover, whence it passes to the motor apparatus of the vehicle, and which again interrupts the current immediately after the passage of the car.

At the present moment three tramway companies are working the Diatto system on a portion of their lines in Paris, viz. the East-Paris, the West-Paris, and the Vanves-Paris Co. and its extensions. The electrical energy is furnished to the tramways of the latter company by a special generating station. Those of the East-Paris and West-Paris Cos. receive theirs from stations at Vitry and Issy-les-Moulineaux, where triphase high-tension current at 3000 volts is generated, being transformed down in sub-stations to a continuous current at 500 volts for actual use on the line. Throughout the entire extent of these companies' lines the mean daily pressure is 500 volts. In the sections where the cable is underground, the current being distributed to surface-contacts as above indicated, the tension of the current at the surface-contact cannot in any case be higher than 500 volts.

In the Diatto, as in the Claret-Vuillemier system, the mechanism, though appearing relatively simple, is in reality complicated, and is subject to disturbance by many causes. To insure the systems acting well, and to prevent such accidents as might result from a tension of 500 volts, it is essential that the communication established between the live cable and the surface-contact at the moment when the vehicle is passing should be interrupted immediately thereafter.

Every precaution appeared to have been taken to ensure these conditions being realised, and the Diatto system was regarded as uniting all the essentials for successful working. This has been the case, it would appear, in some towns, but in Paris the experience has been less favourable. Scarcely had the vehicles begun to run when a perfect chapter of accidents, involving men and horses, was recorded. In six months nearly 100 occurred ; during a single month more than fifty were reported. As regards horses, the greater number of cases resulted only in severe shaking, without serious consequences, though a number of animals were killed on the spot.

The circumstances under which these fatalities to horses occurred necessarily aroused public opinion. The Municipal Council of Paris,

the Prefect of Police, and the Minister of Public Works took up the question. Being consulted by the Ministry, the Governmental Railway Committee enquired into and determined the cause of the accidents. Without doubt these accidents were almost always due to the surface-contacts being permanently electrified, but the Committee indentified five different causes, viz. :—

- (1). Mechanical imperfections in the surface-contacts.
- (2). Insufficient insulation of the surface-contacts.
- (3). Injurious action of the rubbing-contacts at the back of the cars.
- (4). The deposition of soot on the interior of the surface-contacts, and lastly,
- (5). Defective insulation of the cables.

The orders given to the companies by the Prefect of Police regarding changes required in the lines equipped on the Diatto system have been carried out, but, although they have become rarer, accidents have not altogether ceased.

The effects which the current produces on a horse, when one of the animal's feet touches a charged surface-contact, depend principally on the energy of the current, on the dryness or moisture of the roadway, on the more or less intimate apposition between the horse's feet and the surface-contact, and on the length of time during which the two remain touching. When the shock results in the animal falling the effects may vary greatly, depending on whether the animal falls on the surface-contact itself or at some distance from it.

In the great majority of cases the electric shock produced in horses as above described is not of special gravity. Even when the animal falls it seldom shows any serious disturbance afterwards. But sometimes the shock causes instantaneous death or nervous disorders, particularly signs of cerebral depression. In certain cases more or less extensive paresis or paralysis follows. I here recapitulate three cases of the kind reported to me by veterinary surgeons. A horse, previously in perfect health, showed after receiving a shock loss of appetite, depression, and chronic affection of the brain. In another, the shock produced dulness and depression, lasting for more than a month. In still another, symptoms of weakness, loss of appetite, and torpidity continued for a week after the accident. These symptoms disappeared, but were followed by hemiparesis of the left side.

The shock is particularly violent if the ground is moist, if salt has been thrown on the road to melt snow, or if at the moment when one of the feet touches the live surface-contact (in communication with the positive pole of the dynamo) another foot touches one of the rails in communication with the negative pole.

In consequence of the high conductivity of the animal's shoes and of the nails, which penetrate far into the horn, as well as of the rail on which one of these shoes rests, the effects of the shock so produced may be fatal. Nevertheless, experiments, to which I shall return, have proved that the horse is not by any means so sensitive to the action of electric shocks as a number of authors have suggested. These experiments show that the shock produced by a current of 500 volts does not always cause the animal to fall, and does not usually produce any grave disturbance provided it lasts no more than

a few moments, particularly if the feet are not moistened, and if by a sudden forward or sideward movement the animal at once breaks the current. The injurious effects are more serious in proportion to the time during which the current continues to pass through the animal's body. If, therefore, the horse falls on the charged surface-contact and remains touching it, the cardiac and nervous disturbance due to shock usually prove fatal. Horses at liberty or merely led in hand rarely receive fatal shocks, because they can, with a single bound, break contact between themselves and the line. On the other hand, animals in heavy draft—horses pulling heavily-loaded buses or tram-cars uphill, for instance—can only move at a slow pace, and therefore plant the feet more slowly and have less liberty of movement. They are the most seriously affected by shocks. Shaft horses, being confined between two shafts and unable to move sideways, often fall dead on the surface-contact itself.

The following, from a report by M. Rossignol, summarises the experiments made at the Electric Generating Station of Saint-Mandé, by a Commission of the Society of Practical Veterinary Medicine.

On the 5th March 1901 this Commission experimented on two horses. In one of the courts of the generating station M. Barry had had constructed an arrangement representing the charged contact plates of the Diatto system. By placing the first horse with one front or one hind foot resting flat on a surface-contact, it was found that a continuous current of 550 to 700 volts only produced more or less violent shock. The animal reared up or its legs collapsed, but as soon as the foot was withdrawn from the plate the symptoms ceased. The second horse showed very similar results.

Each pair of contacts having been covered with a plate of sheet-iron to enlarge their surface, a new trial was made with the first horse, the front feet resting on one of the plates, the hind on the other. A continuous current of 550 volts immediately caused the animal to fall on its side, but it was able to rise again, and after making several plunges rapidly recovered. It was then wetted all over, water being applied to the limbs, belly, and quarters, and having again been placed on the sheets of iron, was submitted to the action of a continuous current of 550 volts. This time the shock was fatal; the horse fell, its limbs extended, its muscles contracted, and with the abdomen, sternum, and lower part of the head in contact with the ground. Then it turned over on the right side and died at the end of a few minutes. The body showed traces of burning at the points which had been in contact with the sheet iron.

The second horse having been placed as above indicated, and subjected to a continuous current of 550 volts, experienced a violent shock and fell down. Nevertheless it was able to rise again, and a fresh application of the current was required to kill it.

On the 23rd March further experiments were made with two horses, the arrangements being slightly different from those in the first series. The first horse was placed so that each of its front feet rested on one of the sheets of iron. A current of 100 volts produced a slight shock and sudden flexion of the knees. Currents of 200 to 400 volts caused the animal to fall on its knees, and produced marked acceleration of the heart's action and of the respiration. At 400 volts

the animal fell on its knees and then on the left side, but rose again in a few moments. A current of 550 volts produced a strong shock and the animal fell, but was able to rise again in a few seconds. When the limbs and belly were moistened the shock was more violent, but not fatal. The hind feet were then placed on the plates of sheet iron, and the animal received a current of 550 volts. It fell on its left side, the croup on the positive plate, the chest on the negative. It made violent and repeated struggles, showed generalised convulsions and grave disturbance of respiration and circulation. It succeeded in altering its position, struggled, and attempted to rise, but the hind limbs were paralysed. The croup having been brought in contact with the positive plate, death rapidly followed.

The second horse, placed in the same position as the first, with one front foot on each of the surface-contacts, received a current of 550 volts. The shock was violent, but the animal did not fall. A second attempt caused the horse to fall on its knees and then on its side; the body muscles were contracted, the breathing and circulation very rapid. Its fall having broken the circuit, the horse was able to rise again. It showed a few superficial burns at the points of contact with the plates. The experiments were then stopped. This horse was kept and placed under observation for several weeks. It showed neither paralysis, lameness, nor any after disturbance.

The principal results of these experiments may be summarised as follows:—

(1). A current of 100 volts produces slight shock and sudden flexion of the knees.

(2). Currents of 200 to 400 volts may cause the animal to fall.

(3). A sudden shock by a current of 550, 600, or even 700 volts, provided it be of very short duration, does not usually cause death or even grave and permanent consequences.

(4). To produce a fatal shock with a current of 500 to 550 volts the action must be prolonged; death does not usually follow until the end of several minutes.

The last number of the *Bulletin* of the Society of Veterinary Science of Lyons contains a report by M. Arloing of similar experiments made there during the month of July 1901, on a section of the electrical line completed for that purpose by M. Roedt, engineer of the Diatto Co. This portion of line consists of a single surface-contact between two rails, the parts being placed at the normal distances. The surface-contact was so arranged as to be constantly charged when the circuit was closed. By means of a voltmeter it was first shown that the current passed freely not only from the surface-contact to the rails but to the different parts of the pavement between the rails and to a certain distance beyond this, especially when the ground was wet. In order that the horse's body may be traversed by the current, it is therefore sufficient that one of its feet should come in contact with the charged surface-contact. Its three other limbs then form negative poles by which the current escapes.

The first animal was a horse weighing 770 lbs., and shod on all four feet. This animal was led on to the line, the pavement being dry. The left front foot was placed on the surface-contact, the right hind on a rail, the two others on the ground; the circuit was then suddenly closed, the tension of the current being 500 volts. The

horse made a slight jump with all four limbs, and fell beyond the contact plate. It was in a state of great excitement. It at once rose, snorted, passed fæces, and took a number of deep breaths, the nostrils being widely dilated. These symptoms completely disappeared in five minutes.

The animal, having been brought back to the road and placed as before, received a shock of 500 volts. It fell to the left; it was placed on and kept lying across the line, the left side of the chest being on the surface-contact plate, the hind limbs resting on the left rail, the front limbs and the head on the right rail. The principal symptoms noted were convulsions, sometimes tonic, sometimes clonic, of the different groups of muscles, a smell of burnt hair, emanating from the portion of the body in contact with the surface-contact, retraction of the eyes into the orbits, dilation of the nostrils; finally, muscular relaxation, opening of the mouth, and evacuation of urine. Death followed at the end of three and a half minutes, during which the tension of the current varied between 400 and 500 volts.

For the second experiment the ground was covered with planks, above which the rails and surface-contact plate projected slightly, the conditions being similar to those in roads paved with wood blocks. During the four experiments the current varied between 500 and 525 volts.

The horse weighed about 740 lbs. Its temperature was 37.4° C. It was placed obliquely across the line, the left front foot on the contact plate, the left hind on the corresponding rail, the others on the wooden plank between the rails. Under the action of the current the animal made a leap and fell on the right side. On rising it was excited, but the symptoms rapidly disappeared.

In two succeeding experiments, with currents of 500 and 525 volts (the animal being placed as on the previous occasion) the shock was less violent and the horse did not fall; it seemed, in fact, as though it had to some extent become accustomed to the electrical discharge.

The planks having been removed and the ground moistened with water in order to increase its conductivity, a fourth attempt was made, and it was agreed that if the horse fell it should be kept over the surface-contact. It was placed between the rails, the left front foot on the surface-contact, the others on the ground, and the circuit of 500 volts was suddenly closed. The animal flexed its legs, fell on the left side, and was drawn by assistants into contact with the charged plate. It immediately showed symptoms like those of the first subject: tonic convulsions of the muscles of the limbs, trunk, and neck, retraction of the eyes into the orbits, dilatation of the nostrils, and very pronounced contraction of the facial muscles. In sixty to eighty seconds the tonic convulsions were replaced by clonic convulsions; a smell of burnt hair was noticeable, the head and trunk became covered with sweat, and death occurred in a little less than two minutes. The rectal temperature was then 38.2° C.

The other experiment consisted in exposing a mule to the action of a current of 500 volts, and keeping it alive in order to study the delayed effects of the electric shock. The animal was placed between the rails, the left front foot on the contact plate, the others resting on the wet soil. Under the action of the shock produced by the closure of the circuit, the animal flexed its limbs and fell across the rails, the

right side of the chest resting on the surface-contact. It was kept there for twenty seconds, during which time it showed great excitement and perspired freely. The current was then cut off. The mule remained lying on the ground for five minutes ; then with a little assistance it rose. The respiration and circulation, though still rather rapid, gradually returned to normal. Although towards the end of the experiment a slight odour of singed hair had been noted, no trace of burning could be seen.

The mule walked the four kilometres between the place where the experiments were made and the Veterinary College without showing anything unusual. It was kept under observation for four days. At first it had little appetite, but it soon returned to its normal condition and showed no disturbance of the principal functions.

The chief conclusions deduced from these experiments were as follows :—

Currents of high voltage only seem to produce death when passed through the animal's body for a period of sixty to eighty seconds. It is necessary that during this time some part of the animal's body should remain in contact with the electrified plate.

A horse killed by the current seems always to show a skin-burn extending to a greater or less depth into the subjacent tissues.

Horses recover easily and rapidly from the effects of a current of 500 volts continued for twenty seconds.

The dangers inherent to the presence of a charged surface-contact plate are reduced to a minimum when the street is paved with wood and is dry.

In Paris horses have often received fatal shocks from tram lines using a current of 500 volts simply by stepping on a charged contact plate, without having fallen on the plate and without showing any trace of burning on *post-mortem* examination. Those of our confrères who have been consulted in connection with fatalities of this kind explain them by the wet condition of the road and by the general state of health of the subjects ; that is to say, by the fatigue, great excitement, over-exertion, etc., of the animals at the moment when they have been subjected to the action of the current.

* Experiments recently made on the action of electric currents on small animals have thrown light on the mode by which death is brought about. Whatever the form of death, such currents kill either by direct action, at the same time producing electrolytic effects, or by reflex action, in consequence of inhibitory impulses resulting from irritation of nervous centres. When death is about to occur the animal shows violent and generalised convulsions ; respiration and circulation, usually accelerated at first, are soon suspended ; the mucous membranes become pale, the pupils dilate, urine may be passed ; one or more deep inspirations are made, and death follows. Arrest of respiration is not usually the immediate cause. Currents of medium intensity (500 to 600 volts) seldom produce such severe effects on the respiratory centre as finally to stop respiration. They act specially on the heart, the ventricular walls of which show fibrillar trembling and cease to contract ; the heart-chambers become dilated with blood, and death follows from cardiac paralysis.

When death follows immediately on the shock it is due to simultaneous paralysis of the heart and respiratory centre. Paralysis of

scen hitherto have been burns due to the horse lying on the charged surface-contact on which it originally fell. As stated above, when the horse is moving rapidly or is comparatively free at the moment of touching the charged surface-contact, it seldom suffers more than a somewhat intense shock. On the other hand, slowly moving horses and these confined in the shafts of heavy vehicles often fall, and may thus bring some portion of the body over the charged surface-contact. It has been shown above that such a fall on a charged surface-contact is generally fatal. It is not invariably so, however, provided the horse is removed in time, but in such case the region which has touched the contact-plate is the seat of a burn proportionate in size and depth to the time during which the skin and plate have been in contact. If the horse whilst down struggles violently, parts of its body may successively come in touch with the charged surface-contact, in which case they show burns of varying severity. Several horses burned in this way showed lesions of so grave a character as to necessitate slaughter.

After falls following on violent electric shocks, nervous disorders like coma, stupor, and nervous twitchings indicating profound cerebral disturbance, have been noted, but these disappeared in time. In others, lameness, localised paralysis, paralysis of both hind limbs or of the hind and front limbs of one side, have been noted. These nervous symptoms are without doubt due, in some cases at least, to hæmorrhages in the nervous centres resulting from the action of the electric current.

The only constant lesions found on *post-mortem* examination of horses killed by electricity—the lesions truly produced by the current—resemble those of asphyxia, being of a congestive and hæmorrhagic character, but usually somewhat discrete and localised in the intestine, pleura, and lung, or, in a less degree, in the serous membrane of the heart and in the nervous centres. The gross changes described after certain *post-mortem* examinations were the result of physiological congestion of certain organs at the moment when the horse received the shock, or were the result of *post-mortem* decomposition.

It is quite true, as has been remarked, that on the surface-contact electric tramlines horses are particularly liable to accidents which are usually ascribed to the electric shock, but in the production of which shock has played no part. In consequence of the gradual wearing of the road the surface-contacts project more or less above the common level, forming a series of mechanical obstacles which are a continual menace to horses. Horses stumble on these and fall, and thus arise claims which in the absence of precise information are very difficult to assess. For whether the horse has fallen in consequence of electric shock, of slipping, or of its foot striking against an obstacle like the projecting surface-contact, the injuries which follow show precisely similar physical characters. The existence of burns is the only evidence on which we are absolutely justified in ascribing the accident to electricity.

resulted from sub-pleural and intrapulmonary hæmorrhage. The heart showed nothing abnormal. In the nervous centres there was no hæmorrhage; only a little injection of the vessels of the brain.

The *post-mortem* examination of the two horses killed in the Lyons experiments revealed the following lesions:—In the first horse, examined ten minutes after death, the skin of the left hypochondriac region was burned; the subcutaneous connective tissue below it was injected and showed hæmorrhagic points. The pleura was yellowish and very hot to the touch; the lung burning hot and dry on the surface; section of the subcutaneous veins led to the escape of reddish laky blood, which coagulated slowly or not at all; there was congestion of both lungs, with hæmorrhagic subpleural effusions, the largest the size of a sixpence; the right cavities of the heart were distended with blood; the right ventricle was soft and flabby, the left ventricle hard and resistant to the touch. No change in the endocardium. In the abdomen the intestinal branches of the portal vein were distended; certain points in the walls of the small intestine and a part of the double colon were congested. There were traces of burning on the anterior surface of the left lobe of the liver, the tissue being yellowish and showing hæmorrhagic spots. The anterior part of the left kidney showed similar changes. The surface of the brain and the floor of the fourth ventricle were slightly congested.

The second horse, examined seven and a half hours after death, which occurred on a very warm day, showed a degree of putrefaction such as would have been expected. There were slight traces of burning of the skin and of the more superficial of the subjacent tissues; congestion of both lungs, especially of the right; subpleural ecchymoses, most marked opposite the anterior lobes; slight congestion of the mucous membrane of the trachea and larynx; sub-peritoneal hæmorrhagic effusions along the colic and cæcal veins and on the floating colon; the small intestine was of a bluish-red colour, and showed numerous branching hæmorrhages. There was slight congestion of the floor of the fourth ventricle.

M. Arloing concludes that the above *post-mortem* examinations have not revealed any lesions characteristic of death by electric shock, but that the congestive changes found here and there in the intestine, lung, and connective tissue, and the colour of the blood, assume special importance when found in a horse which has died whilst passing over an electric tramway line, particularly if they are accompanied by traces of burning.

Viewed collectively, the lesions shown are those of asphyxia; and if, apart from the burning, there is nothing of an absolutely unequivocal character to point to, yet the *post-mortem* appearances acquire a special significance when the horse has died within a few minutes of its fall. To state the case otherwise, when death follows suddenly, apart from the action of the electric current, one expects to find the lesions which have determined it—injury of the brain, rupture of the heart or of one of the large thoracic or abdominal vessels.

To sum up, horses which have received an electric shock seldom show other injuries than those produced by the fall; but continued contact with charged surfaces may produce grave and more or less permanent lesions of varying characters. Those most commonly

Nedrigailov¹ and Mirto Domenico² by injecting with human ascitic liquid obtained sera which precipitated human albumen.

Finally, Hausner,³ Butza, Linossier and Lemoine,⁴ Minovici, Strube,⁵ Michaelis, Rostoski, Umber and Falloise,⁶ published interesting researches on the preparation and properties of precipitating sera.

This historical survey suffices to show that it is possible to obtain sera which precipitate, *in vitro*, albuminoid substances by the appropriate treatment of various animals with these substances. The precipitating property of these various sera is precisely the result of the treatment carried out. It is, in fact, easy to observe that the serum of normal animals which have not been treated in any way do not possess the power of precipitating solutions of the various albuminoid substances; one may thus formulate the following law:—

If on several occasions one inoculates into an animal A an albuminoid material (blood serum, semen, ascitic liquid) coming from an animal of a different species B, the serum of the animal A acquires the property of precipitating, *in vitro*, albuminoid solutions derived from the species B.⁷

Sera thus obtained have therefore been properly termed precipitating sera, and we shall see later the important practical purpose to which they may be applied.

The precipitating sera, whatever they may be, present one common character—viz., that their properties are not altered by exposure for one hour to a temperature of 55° C. That effect is produced only after heating to 70°. In this they are distinguished from the cytolytic, bacteriolytic, or toxic sera, which are very sensitive to heat. On the other hand, in this point they resembled the agglutinating, anti-toxic, or anti-infectious sera. It has been proposed to give to their active ingredient, which is precipitated by sulphate of magnesia, the name of coagulin, but this has been rejected in favour of the term precipitin, which is more exact, since the turbidity which is produced in albuminous solutions by the addition of the corresponding precipitating serum is the result of the precipitation of albumen and not of coagulation of that substance.

PREPARATION OF PRECIPITATING SERA.

As we have already seen, it is an easy matter to obtain precipitating sera. It is interesting, however, in view of the practical importance which attaches to the preparation of these sera, to consider the methods by which one may very rapidly obtain a serum as active as possible.

According to the observations of some authors, the choice of the animal which is to furnish the serum appears to be a matter of great importance, for, as Bordet discovered, the production of an active

¹ Nedrigailov: "Vratch," 1901, p. 857.

² Mirto (Domenico): "Reforma Medica," 1901, p. 857.

³ Hausner: "Nevrologitchesski Vestnik," 1901, p. 845.

⁴ Linossier and Lemoine: "C. R. Société de biologie," 1902, p. 85, 276, 320, 369, 415.

⁵ Strube: "Deutsche medic. Wochenschrift," 12th June 1902.

⁶ Falloise: Contribution à l'étude des sérums précipitants. "Annales de l'Institut Pasteur," 1902, p. 833.

⁷ Kowavski ("Deutsche medic. Wochenschrift," 1901) has obtained sera which precipitate vegetable albumens.

serum seems to be directly related to the strength of the impression produced by the inoculations in the experimental animal.

A priori it is therefore advisable to choose for the preparation of a given precipitating serum an animal belonging to a species as different as possible from the one which will furnish the albuminoid material used in the treatment.

For example, in order to obtain a serum that will precipitate the albumens of the sheep, one will treat with these substances a rabbit or a dog in preference to a goat. Uhlenhuth considered the rabbit the best subject for the production of precipitating sera of various sorts, but Hausner preferred the dog for that purpose.

The choice of the animal to produce the serum having been made, what is the best substance to employ for the inoculations?

The works of Bordet, Tchistowitch, and Nolf have shown that the red corpuscles of the blood, when inoculated into animals, do not play any rôle in the formation of the precipitating substances. On the contrary, the essential rôle in this production devolves on the plasma or on the serum. It is, therefore, preferable to use in the preparation of the animals blood serum rather than blood itself.

When the object is to prepare serum which will precipitate albuminoid materials of animal origin, it is easy to obtain large quantities of serum by bleeding; but when one wishes to obtain serum that will precipitate human albuminoids, since it is rather difficult to procure human blood by repeated bleeding, one may utilise ascitic liquid or pleural effusion, or even blood from the placenta, or that which has been taken from the dead body.

These different organic products contain a mixture of various albuminoid materials, which are, as yet, badly known. Among these substances there are some which are readily precipitated by saturation with sulphate of magnesia; these are the so-called globulins. Others, on the contrary, remain in solution; these are the serins. Nolf¹ has proved that the precipitating property appears sooner in animals treated by injections of globulin than in those treated with serin.

There is, however, no inconvenience in employing ordinary serum, and since it is simpler to treat animals in that way it does not appear to us to have any interest to use, with this object, albumens which have been purified or separated from blood serum by precipitation with sulphate of magnesia or ammonia.

Apart from the choice of the animal and of the substance with which it is to be inoculated, the value of a precipitating serum depends on the quality of the substance inoculated, the place at which the injections are made, and the duration of the treatment. Intravenous and intra-peritoneal inoculations give a more active serum than subcutaneous inoculations.

On account of its simplicity, intra-peritoneal inoculation of tepid serum is to be preferred. In our own hands this has given the best results. Moreover, whatever be the method of inoculation adopted, it is a good plan to raise the sera intended for treatment to a temperature of 55° in order to destroy their toxicity.

The precipitating power of a serum furnished by a given animal increases in proportion to the length of the treatment. Thus, from

¹ Nolf, *loc. cit.*

rabbits treated with human blood every day for six days, Stern obtained a serum which was active in the strength of 1 per 1000. After several months of treatment this serum acted in the proportion of 1 in 50,000. Moreover, it is important to repeat injections of the albuminoid substances chosen as often as possible. Provided the health of the subject permits, the inoculations ought to be repeated every two days at least.

The serum may be collected from a treated animal the day after the last injection. However, it is better to wait five or six days, and always to bleed the animals in the fasting state. One thus obtains a serum which is absolutely limpid and devoid of bacteria, and which therefore keeps well when it is collected with aseptic precautions.

When one interrupts the injections of the albuminoid substance into the animal intended to furnish the serum, the precipitating power of the serum falls; and, according to Steube, two months after cessation of the treatment, rabbits' serum which was originally active in the strength of 1 per 1000 has entirely lost its precipitating power. Moreover, the same author has observed that when the treatment of such animals is resumed they soon furnish a serum that is much more active than that which was obtained after the first period of treatment.¹

Precipitating sera which have been collected pure and kept cool and protected from light will retain their precipitating properties for several weeks. Rapid desiccation of the sera at a low temperature *in vacuo* preserves their properties for a much longer time.

RESEARCHES REGARDING PRECIPITATING POWER.

In order to put in evidence the existence of precipitating power in a serum prepared in the manner described above, it suffices to add to a given volume of the albuminous solution employed in the treatment an equal volume of the prepared serum. As the precipitated albumen which forms is soluble in excess of the precipitable solution, it is better to increase the proportion of the precipitating serum. If one employs albuminous dilutions it is always better to make these dilutions in physiological salt solution (.8 per cent.). It is important to utilise in such researches only liquids that are very limpid.

When one endeavours to dissolve organic albuminous substances, such as blood, semen, or sputum, in physiological salt solution, one always obtains a turbid mixture. It is well to clarify this by repeated filtration through paper before submitting it to the action of precipitating serum.

Immediately after the substances have been mixed the original limpidity of the two liquids appears to persist, but after some minutes the liquid becomes opalescent or milky, and gradually loses its limpidity. After some hours the limpidity resolves itself into a more or less abundant flocculent precipitate. In every case the reaction is extremely distinct, and it is always obtainable when a given serum is mixed with an albuminous solution of the same animal origin as that

¹ When one employs rabbits in order to produce serum, and the treatment has been brought to a termination, it is better to sacrifice them by extracting all the blood possible from the carotid.

which was employed in the treatment of the animal furnishing this serum.

The temperature does not seem to exercise any marked influence on the reaction. According to Linossier and Lemoine,¹ the flocculent deposit is not readily obtained at 0°, and the reaction is most rapid at about 35°. I have always operated at the temperature of the laboratory (15° to 20°), and in every case I have obtained excellent results. It is best to operate with a neutral or slight alkaline medium.

The delicacy of the reaction is altogether remarkable. Uhlenhuth,² for example, prepared a serum which precipitated the albumen of cows' milk, and found that a few drops of it added to a dilution of 1 per 100,000 was active, whereas, according to the same author, the chemical reactions were not discernible in weaker solutions than 1 per 1000. Hausner³ obtained for the precipitation of human blood a serum so active that when a dose of $\frac{1}{4}$ cc. was added it produced a distinct precipitate in the liquid obtained by diluting a drop of blood in 15 cc. of water. Linossier and Lemoine⁴ prepared a serum capable of revealing the presence of albumen in a dilution of 1 cc. of serum in five litres of water, and which could thus put in evidence amounts of albumen less than the hundredth part of a milligramme. On many occasions, by employing the anti-albuminous serum described by Leclainche and myself, I have been able to detect in human urine traces of albumen which were not recognisable by the chemical tests.

SPECIFICITY OF PRECIPITATING SERA.

The specific nature of the reaction furnished by precipitating sera on different albumens found in the same animal species, and on albuminoids furnished by subjects belonging to different species, is deserving of study.

With regard to the specificity of sera towards different albuminoid substances of the same species, opinions are divided. Thus, Nolf, Leblanc, and Michaelis prepared sera which precipitated blood serum and solutions of globulin, but not solutions of serin; while Linossier, Lemoine, Rostoski, and Falloise prepare animals by injecting them with globulin or serin. Uhlenhuth showed that the active precipitant of human serum is equally active in precipitating the other albuminous liquids of the organism, such as semen, purulent sputum, etc.

If for a moment we admit that it is possible to distinguish different human albumens by the employment of precipitating sera, a more profound examination of the matter has shown that in some cases one cannot count on such a result; and we agree with the authors who consider that the precipitating sera cannot be employed to distinguish between the albumens of the same animal species.

The results furnished by the precipitating sera are much more precise when the object is to distinguish between the albumens from animals belonging to different species.

Almost all the authors who have studied the precipitating sera ad-

¹ Linossier and Lemoine. Loc. cit.

² Uhlenhuth. "Deutsche medic. Wochenschr." 15 November, 1900.

³ Hausner. Loc. cit.

⁴ Linossier et Lemoine. "C.R. Soc. de biologie." 1902, p. 87.

mit with Tchistowitch, Bordet, and Uhlenhuth that these reactions are specific. In other words, the serum of a rabbit treated by repeated injections of blood or serum from the human subject precipitates only dilutions of human blood and serum, to the exclusion of serum from any of the lower animals. Leclainche and I have shown that the serum of rabbits inoculated systematically with human albuminous human urine precipitates only human urine, and leaves absolutely limpid albuminous urine from the various domesticated animals.

This specificity is very generally admitted. It has been observed, however, that a serum prepared to precipitate the albumens of a given species also precipitates, but to a slighter degree, the albumens from a species nearly related in the zoological scale. Thus, Myers has shown that the serum which precipitates albumen of the egg of the common fowl also precipitates that of the duck, and Uhlenhuth observed that serum active for fowl's serum also precipitated that of the pigeon. Again, Stern found that the precipitant active for human serum also acts on the serum of certain monkeys. However, all these authors recognised that a serum very active on human blood, for example, does not precipitate the serum of any of the domesticated animals, and that is the opinion generally accepted.

Some months ago MM. Linossier and Lemoine¹ observed "that the same precipitin may act on a very large number of different sera."

It is well to notice that these authors in their experiments added to not less than ten volumes of precipitating serum one volume of serum from different animals. These, however, are very special technical conditions, and the results change completely when, as I have often done, one mixes equal volumes of the precipitating serum and of the substance to be precipitated. When one works in this way the reactions are invariably specific.

APPLICATIONS OF THE PRECIPITATING SERA.

From what has just been said it is easy to foresee the useful practical applications to which the precipitating sera may be put.

When used in certain conditions they constitute excellent reagents to indicate the species which has furnished a given organic substance. Uhlenhuth was the first to propose to use precipitating sera for the diagnosis of blood stains, which are so important in legal medicine, and he made known the method in which this test ought to be carried out. It suffices to dissolve the blood stain which has to be examined, present on an article of clothing or an instrument, in a solution of 8 per cent. of chloride of sodium, and then to filter several times until an absolutely limpid liquid is obtained. To this solution one adds some drops of serum from a rabbit or guinea-pig treated with human blood. If the suspected stain is one of human blood a precipitate appears in the mixture, but if it remains limpid the blood must have come from some of the lower animals, and the precise species may be easily determined by testing the solution with sera which precipitate the albumens of the different animal species. According to Uhlenhuth the reaction may even be obtained with human blood which has been allowed to putrefy for several months.

¹ Linossier et Lemoine. Loc. cit.

This method also gives excellent results in the determination of spots of semen.

If one operates with sera of which the activity has been previously determined, and takes care to prepare control tubes, one may accept without reserve and with every confidence the indications afforded by the experiment.

Various authors, notably Wassermann, Schütze, Nedrigarlov, and Ziemke, have confirmed the value of Uhlenhuth's method, and Minovici declares it to be superior to those previously employed in legal medicine for the diagnosis of blood stains.

The sera that precipitate milk, which were first studied by Bordet, have been put to an interesting practical application by A. Schütze,¹ who showed that the serum of rabbits inoculated with cow's milk precipitates only the albumens of such milk, to the exclusion of all others. In the same way, rabbits treated with the milk of the goat furnish serum which precipitates only goat's milk. These sera may thus be utilised as very useful tests by cheese-makers who employ in their industry the milk of ewes. In view of the higher commercial value of the milk of the ewe, it is frequently adulterated with the less valuable milk of the cow. In the future it will therefore be possible, by testing the milk with different precipitating sera, to discover these frauds, and to determine the nature of the milk introduced into the mixture.

The precipitating sera have received still another valuable application in differentiating different kinds of flesh. This particularly important application will be made the subject of a special study.

Some very interesting biological facts have been brought to light by these precipitating sera. For example, Uhlenhuth has by this method proved that the albumen of the egg of the fowl is different from the blood serum of the fowl, and Kowarski² has been able to distinguish between the different vegetable albumens and to separate them from those of animal origin.

The precipitating sera are not only interesting from the scientific point of view, but also furnish valuable methods of investigation to chemists, and to those employed in legal medicine. Their application to the differentiation of samples of milk and of butcher-meat has appeared to us to justify the preparation of this critical review.

A PECULIAR STAINING REACTION OF THE BLOOD OF ANIMALS DEAD OF ANTHRAX.

By J. M'FADYEAN, Royal Veterinary College, London.

It will hardly be denied by anyone acquainted with the facts that at the present time the majority of ordinary practitioners, whether human or veterinary, do not habitually have recourse to bacteriological methods in the diagnosis of disease, even in those cases in which such methods are alone capable of conducting to a certain conclusion. This is notably the case with regard to anthrax. In this country the evisceration of the carcase of an animal suspected to have

¹ *Zeits. für Hygiene*, 1901, p. 5.

² *Deutsche Med. Wochen*, 1901, p. 442.

died from anthrax is contrary to law, but, even if it were permitted, there are a good many cases in which an ordinary macroscopic *post-mortem* examination of the internal organs would not justify an absolutely confident diagnosis. On the other hand, in the circumstances of every-day practice, there are very few cases in which a competent person cannot by microscopic examination of the blood of the dead animal determine with a near approach to certainty whether it has died from anthrax or not.

In the preceding sentence much turns upon the word competent, and the question arises, who is for this purpose a competent person? The question is not one easy to answer briefly. It may safely be said, however, that a necessary element in the competency is some previous experience in the microscopic examination of anthrax blood. No mere theoretical acquaintance with the matter, and no amount of reading of the recorded experience of others, entitles anyone to consider himself competent to diagnose anthrax by microscopic examination of the blood of the suspected animal. On the other hand, anyone who possesses a microscope of sufficient power, may without much trouble make himself competent to do this. All that is necessary is a little experience in the comparison of properly prepared blood preparations from animals dead of anthrax with similar preparations from animals dead of other diseases.

The question as to what constitutes an efficient microscope for this purpose is also one of considerable importance. It is true that in certain circumstances a very ordinary instrument, capable only of a low magnification, may be quite sufficient—viz., when it is known that the suspected animal is so recently dead that the blood to be selected for examination cannot possibly contain putrefactive bacteria. Unfortunately, in many cases the practitioner is called upon to make a diagnosis without this assurance, and to place reliance upon what is then seen through a microscope of low power exposes the person making the examination to serious risk of error. In this connection a good deal of practical mischief has been done by the recommendation of so-called "pocket microscopes" for the diagnosis of anthrax. Whoever intends to base his diagnosis of this disease on microscopic examination of the blood ought to equip himself with a good modern instrument, provided with a sub-stage condenser and an oil-immersion lens, and capable of magnifying at least 800 diameters.

Assuming that such an instrument is available, the source of the blood to be examined may next be considered. As previously mentioned, the evisceration of the carcase is forbidden, and rightly so, in view alike of the risk to the operator and the certainty of subsequent serious soil contamination should the case prove to be one of anthrax. Fortunately, there is good reason to believe that this restriction in no way hampers the veterinary surgeon in his endeavour to prove by microscopic examination whether the disease is anthrax or not, at least when he is dealing with an ox, sheep, or horse.

In these species anthrax is probably never fatal before the bacilli have become generalised and invaded the blood in large numbers. In the pig, however, it is certain that at the time of death the blood may still be free from the bacilli, or contain them so sparingly as to make microscopic examination of it unreliable. In passing it may be observed that this is not a fact which justifies a complete *post-*

mortem examination when a pig is suspected of having died from anthrax. In most cases of the kind the history that the animal has recently been fed with the raw flesh or organs of some other farm animal unexpectedly found dead, and the fact that swelling of the region of the throat was one of the symptoms exhibited during life, may safely be accepted as satisfactory evidence that the disease is anthrax; but if it is considered necessary to furnish microscopic proof, and blood from a peripheral vein fails to provide this, the œdema of the throat and the lymphatic glands there should be examined before the organs of the chest and abdomen are exposed.

In the other ordinary animals of the farm no dissection beyond that which is necessary to obtain a drop of blood should ever be made until microscopic examination has indicated that the case is not one of anthrax. If the carcase is not very putrid, and the animal has died from anthrax, blood taken from any of the superficial veins will contain the bacilli; and, as is now well recognised, if putrefaction has made great progress it might be impossible any longer to recognise the bacilli in blood taken from the interior of the body. Apart, therefore, from the objections to evisceration previously mentioned, there is a positive disadvantage in selecting the blood for examination from the spleen or the vessels of the abdomen or thorax.

The next point to be considered is the particular stain to be employed for staining the blood in suspected cases of anthrax. Some authors have insisted on the fact that in an ordinary blood film the bacilli of anthrax, when present, may readily be detected although unstained. The fact need not be denied, but it is permissible to question the wisdom of advising that those who are not experts should rely upon the microscopic examination of the unstained blood. The anthrax bacillus need not be deprived by the process of staining of any of its characters valuable for diagnosis; on the contrary, the process of staining may be employed to bring out features in the bacilli that are not visible when they are unstained.

A considerable number of special methods of staining anthrax bacilli have been recommended, most of them being directed to bringing into prominence the relatively thick sheath or envelope with which the bacilli are provided. For that purpose the methods of Johne, Klett, and Olt are useful. In doubtful cases one may also usefully have recourse to Gram's method, in order to discriminate between anthrax bacilli and malignant œdema bacilli present as a *post-mortem* invasion of the blood. Like some of those above mentioned, Gram's method lacks simplicity, and in the hands of the unpractised it may easily lead to error.

The principal purpose of the present note is to call attention to a peculiar staining reaction of the anthrax bacillus, which, so far as the writer is aware, has not previously been described, although it is of a rather striking character, and of great value for the microscopic diagnosis of the disease.

The reaction is exhibited when films of blood, exudate, or tissue juice containing the bacilli are stained with simple aqueous solution of methylene-blue. When applied to the examination of blood, the procedure is as follows:—

On the centre of the upper surface of a clean microscopic slide place a droplet of the blood. The droplet should be of such a volume

that, when placed on the slide, it will cover a surface about the size of the capital letter at the beginning of the preceding sentence. With the platinum or other needle, quickly spread the blood as uniformly as possible until it covers a circle about half an inch in diameter. Set the slide aside, protected from dust, until all appearance of moisture has disappeared from the film. At ordinary temperatures this will not require more than a few minutes. As soon as the film is dry, heat the slide by lowering it, film-side upwards, into the flame of a Bunsen burner or spirit lamp for a second. Repeat this three times, or until the under surface of the glass is a little too hot to be borne by the skin of the palm of the hand. Allow the slide to cool, and then cover the film with 1 per cent. aqueous solution of methylene-blue. After a few seconds pour off the superfluous stain, and wash the slide thoroughly in tap water. Dry the slide by pressing it gently between two layers of bibulous paper, and then more thoroughly by holding it in the current of hot air above the Bunsen flame. Finally, place a drop of Canada balsam on the centre of the stained film, and apply a cover-glass. The preparation is now ready for microscopic examination.

When examined under a sufficient magnification (800 to 1000), the only formed elements visible in any field of the preparation will be the anthrax bacilli and an occasional leucocyte. The nuclei of the latter generally exhibit a greenish-blue tint, while the anthrax rods are stained blue, of which the depth is liable to some variation, depending mainly on the length of time after death that has elapsed before the blood was taken from the carcase. The segmented character of all but the shortest rods will be apparent, although sometimes when the bacilli are deeply stained this is not very distinct. In all this there is nothing peculiar, or special to the method of staining now being described. The peculiarity in the reaction lies in the colour of the amorphous material which is present between and around the bacilli. This material presents itself under the form of coarse or fine granules of a violet or reddish-purple colour, which is in sharp contrast to the tint of the bacilli or cell nuclei, especially with brilliant lamp or gas light. These violet granules differ a good deal in form and size. Sometimes they are very minute, and at other times comparatively coarse. When the bacilli are arranged in clumps and groups the violet material is often in greatest amount in their neighbourhood, and even free-lying bacilli may be surrounded by a thick envelope of the same substance.

The foregoing description applies also to the film preparations of the local oedema, spleen pulp, or juice from the other organs, but the violent reaction, in respect both of the intensity of the colour and the amount of the stained material, is generally best exhibited by blood films. The reaction is constant in anthrax of any of the domesticated animals, as well as in the mouse, guinea-pig, and rabbit. It is obtainable immediately after death, and it may still be detectable when all the anthrax bacilli have undergone dissolution in the unopened carcase.

Finally, in the experience of the writer, the same reaction is never obtainable in animals dead from any other disease or condition than anthrax, no matter whether the examination is made immediately after death or when putrefactive bacteria have invaded the blood.

Although the microscope is necessary for the proper appreciation of the reaction, it is generally possible to tell with tolerable certainty whether the case has been one of anthrax or not by naked-eye inspection of the stained film. When a film of normal blood, or even of blood that contains large numbers of bacteria other than anthrax bacilli, is treated with methylene-blue in the manner before-described, it seldom becomes very deeply stained, and the tint of the parts to which the stain has attached itself is blue or greenish-blue, without any shade of red and purple. On the other hand, an anthrax film similarly treated stains deeply, and there is a distinct trace of red or purple in the film, especially when it is held up to the light. Anyone who will make the comparison between blood-films thus stained from rabbits dead from rabbit septicæmia or fowl-cholera, and similar films from rabbits dead of anthrax, will immediately be struck with the great difference in the macroscopic appearance of the films in the two cases.

Returning for a moment to the method of preparing and staining the films, it is important to notice that what might be thought trivial departures from the directions given above will entirely prevent one from obtaining the reaction. For instance, if the film be fixed with sublimate, formalin, or osmic acid solutions, no trace of the violet reaction will be obtained. Failure will also occur if in fixing the film by dry heat the temperature is allowed to rise as high as 150°C .; 100°C . is sufficient, and it should not be allowed to rise much above that. Again, the film ought not to be very thin. Partly for this reason, and partly because of over-heating in the Bunsen flame, such thin films as are obtained by pressing two cover-glasses together generally fail to show the violet reaction. As an alternative method of fixation the slides or cover-glasses carrying the dried films may be immersed for a few minutes in absolute alcohol or methylated spirit. The stain must not be applied to the hot slide, or heated until the steam begins to rise, as is recommended for some methods of staining, nor must the preparation be washed in alcohol after staining.

It is apparently essential to the production of the reaction that the bacilli shall not be firmly fixed. As is well known, immersion in sublimate, formalin, and osmic acid solutions of sufficient strength not only renders the albuminous film insoluble, but also fixes the red corpuscles in it, so that these preserve their form and position when the film is subsequently stained. On the other hand, when a blood-film is first air-dried and then raised to a dry temperature not exceeding 100°C ., although the albumen in the film is rendered insoluble in water, the red blood corpuscles are in no way fixed. Hence, when such a film is afterwards wetted with a watery medium the red cells undergo instantaneous dissolution and disappear. The same is true for films of which the albumen has been rendered insoluble by alcohol; and, whatever may be the exact explanation, apparently only those methods of preparing the film that leave the red corpuscles unfixed permit one to obtain the violet reaction with methylene-blue. As previously mentioned, dry heat at 150°C . prevents the reaction, but at that temperature the red cells of the blood are completely fixed.

The question now arises, what is the nature of the material in anthrax blood and tissue juices that stains violet with aqueous solution of methylene-blue? There appears to be little room for doubt

that this material is derived from the envelope of the bacilli. As previously stated, and as shown in Plate I., Fig. 2, the violet-stained substance is often mainly found in proximity to the bacilli, rather than at some distance from these; and not infrequently a number of the bacilli are provided with a partial or complete envelope of the same material. Besides, if a film that has been treated in such a way as to bring out this reaction be decolourised and restained by Olt's method, it will be found that the bacilli have been deprived of their envelopes. It is important to mention in this connection that the violet reaction is not obtainable with artificial cultures of the bacilli.

However, the explanation of the reaction is not practically important. The important fact is that such a peculiar reaction is constantly obtainable from anthrax blood. It is not recommended that anyone should rely solely on this test in the examination of the blood of suspected animals, but it is hoped that a knowledge that such a reaction is obtainable in cases of anthrax may prove of some service to those who find that they are occasionally left in doubt by other methods of staining.

DESCRIPTION OF PLATE I.

All the figures are of film-preparations treated in the manner described in the text. Figs. 1 and 2 represent the magnification with a Reichert $\frac{1}{8}$ in. oil-immersion objective and ocular No. 3; in Figs. 3 and 4 the ocular was the same but the lens was a Zeiss $\frac{1}{8}$ in. oil-immersion.

FIG. 1. Blood from the tail of a cow dead of anthrax. The greenish-blue bodies are the nuclei of leucocytes; the anthrax bacilli are stained dark blue, and between them is seen the reddish-purple or violet material.

FIG. 2. From the spleen of a guinea-pig dead of anthrax. The greenish-blue bodies are cell nuclei. The violet-stained material is sparing in amount, and mainly in the neighbourhood of the anthrax bacilli.

FIG. 3. Blood from the heart of a guinea-pig dead of anthrax. The Fig. shows the violet-stained material mainly around the bacilli. The greenish-blue bodies clustered together are fragments of the nucleus of a leucocyte.

FIG. 4. Partially putrid blood from the ear of a sheep dead of anthrax. a. An anthrax rod of which the segmentation is still obscurely recognisable; b. b. and c. c. Anthrax bacilli in various stages of degeneration; d. The last visible vestiges of two anthrax bacilli. All the unlettered bacilli in the figure are putrefactive organisms, the largest (dark blue) being malignant oedema bacilli.

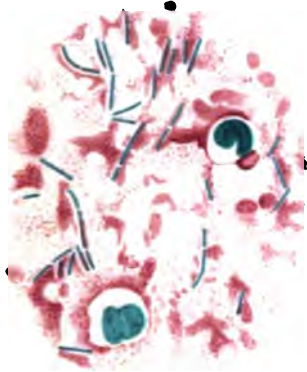
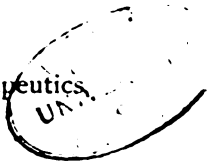


Fig 1



Fig 2

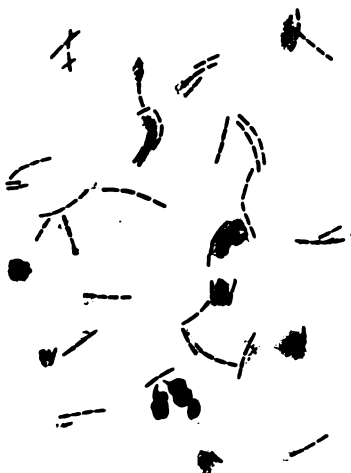
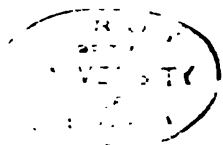


Fig 3



Fig 4



EDITORIAL ARTICLES.

THE BOARD OF AGRICULTURE ON AVIAN TUBERCULOSIS.

FOR the information of owners of poultry the Board of Agriculture has recently published a "leaflet" on the subject of avian tuberculosis. The desire of the Board to disseminate information likely to be of value for the prevention of the contagious diseases of the domesticated animals is praiseworthy, but we fear it must be admitted that the latest effort in this direction will not add to the Board's reputation for accurate knowledge regarding the ailments of poultry.

The leaflet starts with the assertion that tuberculosis "is one of the most common diseases of fowls, turkeys, pheasants, partridges, grouse, pigeons, and other birds." This, it need hardly be said, is a wildly-inaccurate statement. It is unfortunately true that tuberculosis is not a rare disease in the common fowl, but one may without any hesitation affirm that it is not "one of the most common diseases" in any of the other birds mentioned. Indeed, with the possible exception of the turkey, not one of the other species mentioned is often affected with tuberculosis, and in most of them a case of the disease would be deserving of a place in a museum on account of its extreme rarity. No person competent to make a really reliable diagnosis has ever met with a case of tuberculosis in a grouse or partridge, and the disease is equally unknown among pheasants if we exclude those reared in a state of close confinement.

Under the head of "Prevention" the leaflet contains the following curious statement: "The stock at a place affected with liver disease may be divided into the resistant and non-resistant. The breeding tends to be done more from the former than the latter, and this natural process of making the stock stronger would be greatly assisted by the owner burning or deeply burying in lime the birds which have died, and improving the condition of the survivors." We venture to say that the distressed owner of a poultry-yard invaded by tuberculosis will find it a hard task to extract any sensible meaning from this cryptic utterance. It must be inferred that the terms "resistant" and "non-resistant" mean respectively resistant and non-resistant to tuberculosis, and on that assumption one is moved to wonder why the individuals naturally endowed with more than average powers of resistance to tuberculous infection should lay any more eggs than those that are more susceptible to the disease. One can, of course, understand that the fowls that are already affected with tuberculosis will probably lay fewer eggs than those that are still healthy, but we can hardly suppose that the learned person who was commissioned by the Board of Agriculture to instruct the British public through the

medium of this leaflet fell into the mistake of supposing that at any given moment in an affected poultry-yard all the fowls that are still healthy possess higher powers of resistance than those that have contracted the disease. Perhaps the writer of the leaflet had somehow acquired the conviction that in the fowl unusual powers of resistance to tuberculosis are in some obscure way co-related with an unusual degree of fertility. On the other hand, that interpretation of the sentences which we have quoted makes it difficult to understand how the burying or burning of the fowls that have succumbed to tuberculosis can affect this natural tendency to a gradual strengthening of the stock by survival of the individuals that are most resistant. The hen that is already dead is surely powerless to interfere with this tendency even if it is left unburied !

The leaflet continues as follows : " To exterminate the disease, however, something more than that is required. A house should be built with a run in a corner of a field apart altogether from the old poultry-yard, or the system of movable houses, with frequent change of position, may be adopted. Then the strong and healthy birds should be carefully selected and put in the new house ; and, if any of them show the least indication of disease, they should be at once removed and the house disinfected with chloride of lime ($\frac{1}{4}$ lb. to 1 gallon of water), or quicklime, or any other good disinfectant. The resistant birds will in this way be separated from the weaker, and will form a foundation for not only a disease-free but a disease-resisting stock."

We have said above that we hesitated to accuse the writer of the leaflet of having fallen into the error of regarding " non-infected " and " resistant " as equivalent terms, but the paragraph last quoted amply justifies that charge. It is perfectly obvious that in his view a strain of poultry capable of resisting the tuberculous infection can be built up by killing off the already infected fowls whenever one is asked to intervene in an outbreak, and by continuing to breed from those that at the moment of intervention had escaped infection. It would be difficult to formulate a more absurd theory than this. The language in which the author of the leaflet has expressed what he would probably call his ideas may serve to bamboozle the peasant who owns a few fowls, or the average farmer's wife who looks after the poultry-yard, but no one with any pretension to pathological knowledge can fail to see that the paragraph we have quoted is simply nonsensical. It must surely be obvious to the lowest grade of educated intelligence that if the healthy fowls are indebted to their own natural immunity or resistance for their escape from infection, there is no need to separate them from those that are diseased ; and, on the other hand, if they are sufficiently susceptible to make their separation necessary, what reason is there for expecting that by breeding from them one will be able to build up a disease-resisting stock ?

As we remarked at the outset, this leaflet is not likely to enhance

the reputation of the Board of Agriculture for accurate knowledge regarding the diseases of poultry. It is incredible that any of the veterinary officers of the Board can have been the author of it, but it is not easy to understand how it came to be issued without their sanction and approval. In the interests alike of the Board and of the owners of poultry, it is to be hoped that the leaflet will be promptly suppressed.

CANCER RESEARCH.

THE attention of our readers is called to the advertisement appearing elsewhere in this number of the *Journal*, in which members of the veterinary profession are requested to co-operate with the Committee of the Cancer Research Fund, by bringing to the notice of the Superintendent of Cancer Research any cases of cancer encountered in their practices. Live cancerous subjects are specially required for the purposes of the investigation, but recently excised malignant growths will also be gratefully accepted.

Although cancer is a disease much more prevalent in man than among the lower animals, the latter are by no means exempt from it, and it may be safely said that there is no disease of equal gravity regarding the cause of which so little is really known. It is eminently satisfactory that such a powerful body as the Cancer Research Fund has been formed for the express purpose of organising and carrying out a painstaking and exhaustive investigation with regard to the whole subject of malignant growths in man and animals, and we venture to say that veterinary surgeons throughout the Kingdom will be happy to do everything that lies in their power to assist in the inquiry.

It would, of course, be rash to predict that as the result of this investigation the etiology of cancer will soon be made plain, but it is not unreasonable to expect that the inquiry now being organised may materially advance our knowledge of the subject by definitely establishing that a number of the alleged causes of cancer have nothing to do with the production of the disease. Unfortunately, it would be almost impossible to prove or disprove some of the theories of cancer-formation by either the statistical or the experimental line of inquiry, but there is one theory—possibly the most fashionable one at the present time—which will readily admit of being tested by the experimental method of research. We refer to the view that the malignant tumours, and especially the carcinomata, represent the results of tissue irritation by hitherto undetermined microscopic parasites. As is now very generally known, it is held by many that this view has been advanced beyond the stage of a mere theory, since, it is alleged, the causal parasites have been demonstrated in or among the elements of the new growths, and the experimental transmission of cancerous

growths by inoculation has already been realised. There is, however, plenty of room for error in forming conclusions as to the nature of minute stained or unstained particles made visible in the tissues of a tumour; and, in view of the diametrically opposite opinions formed by persons of equal authority regarding the nature of so-called cancer parasites, it is not possible to be very sanguine regarding the results to be obtained by that line of inquiry. The question of the experimental transmission of cancer rests on a more hopeful footing. It is true that here also the evidence already in existence is conflicting, but it is certain that by multiplying experiments it will be possible to determine with an approach to certainty whether any of the common malignant growths are transmissible after the manner of the diseases known to be caused by bacteria and other minute parasites. And in this connection it is fortunate that cancer is not confined to man, for had that been the case it would not have been permissible to attach great importance to the uniform failure of multiplied attempts to transmit human cancer to animals by inoculation or otherwise. But, since cancer does occur naturally in the horse, for example, it ought to be possible experimentally to transmit the disease from one horse to another, if the cause of it is micro-parasite. That is not to say that every such an attempt ought to succeed, but there ought to be a considerable proportion of successes, and the uniform failure of a large series of experiments made with that object would have to be accepted as finally disposing of the theory that cancer is a parasitic disease. On the other hand, should this line of investigation prove that the disease is transmissible experimentally, the result would entitle us to entertain the most sanguine hopes that the discovery of means of curing or preventing the disease would follow. It is for these reasons that the investigations now being begun with regard to animal cancer will be followed with the greatest possible interest.

Reviews.

The Common Colics of the Horse. By H. Caulton Reeks, F.R.C.V.S. 224 pages. London: Ballière, Tindall & Cox, 1902.

THE causation and treatment of colic is a subject of perennial interest to veterinary surgeons, especially to those who find themselves in extensive agricultural districts, where hard work and ignorant treatment render the horse extremely subject to attack. In towns, on the other hand, more intelligent and humane methods have gone far to diminish the liability of equine patients to the group of diseases of which Mr Reeks treats, so that, while that gentleman estimates the percentage of cases of colic in some practices as varying between 50 and 90 per cent. of the total number of cases seen, in many large city practices the percentage of such cases probably does not exceed ten or fifteen.

The earlier portions of the work deal with the surgical anatomy of the abdomen, the methods of examining the patient, and the predisposing and exciting causes of colic, the body of the book being devoted to the various forms of colic, which Mr Reeks considers under an anatomical classification. His chapters on obstruction of the colon and small intestine bring together in an effective form a large mass of hitherto scattered records, and in his treatment of this, as well as in his selections from his own recorded cases, Mr Reeks shows much ability. He is a strong advocate of the stimulant as opposed to the narcotic treatment of obstructed colon, and from the results adduced certainly has reasons for his preference. His chapter on the surgical treatment of intestinal obstructions reproduces in an interesting manner the main outlines of the paper read before the National Veterinary Association in 1895 by Professor Macqueen. The later chapters deal with intestinal tympany, enteritis, and superpurgation. Not the least valuable portion of the book is an appendix by Mr E. Wightman Bell, F.C.S., on the chemical composition of certain foods.

A Treatise on Surgical Therapeutics of Domestic Animals. By P. J. Cadiot and J. Aluny. Translated by Prof. A. Liautard. Vol. I., Part III., pp. 196 to 323. New York: William R. Jenkins, 1902.

THE excellent *Traité de Thérapeutique Générale Vétérinaire* of the above authors is well known and favourably received in all French-speaking countries. It therefore seems regrettable that in arranging for a translation the publishers should have chosen a gentleman who, though most amiable and worthy of respect, possesses an ineradicable disability for the task in question, in that he seems totally incapable of expressing himself in plain English. We have no doubt that had Professor Liautard undertaken to translate an English work into French, his native tongue, he would have acquitted himself with distinction. We only regret that we cannot speak in equally assured terms of his efforts in the opposite direction. The portion of the original book represented by this "Part" deals with (1) surgical diseased conditions common to all tissues, (2) diseased conditions special to each tissue, and (3) diseases of the limbs. Professor Liautard begins his remarks on "Granulations" as follows:—

"When they are situated in regions where there are frequent motions (those upon which the harness is) or on the tissues of the foot, they interfere with work and even prevent it, or impose a more or less active interference, such as the removal of the innodular part, the thinning of the new hoof, or neurotomy. When the pain seems to be due to the adherence of the cicatrix to the tissues underneath, this must be divided subcutaneously, and a sound adherence prevented by passive movements applied on the cicatricial plate. This operation is, however, seldom successful" (p. 196).

We are scarcely surprised. Even supposing the pain to have been subcutaneously divided and the cicatricial plate sufficiently agitated, a successful result would hardly be looked for—by an Englishman. In France they arrange these things better.

"Actinomycetes," we learn, penetrate through "tegumentary solutions of continuity" and "exhibit the following features: Hard tumours, and immediate inflammatory indurations and true neoplasms, hollowed with numerous fistulæ" (p. 202). "In animals, as in man, tumours may appear on parts the most different. . . . The testicle . . . is most often affected. The ectopia of this gland predisposes it to neoplastic degenerations" (p. 215). "Serious and even fatal accidents may occur from bloody exeresis of a

benign and painless neoplasm. . . . During the last few years the prophylaxis of cancer has been much talked about ; but we have seen that, aside from heredity, there are no positive etiological data to admit it" (p. 218).

The Professor has evidently had a wide and peculiar acquaintance with cancer, for he declares (p. 219) that "numerous are the mammary neoplasms of sluts that demand such extensive removals," and a little further on that certain forms "recidivate immediately" after operation.

His definitions of "stickfasts" (suggestive of somebody's widely advertised adhesive) and "cores" are worthy of attention. "The . . . surfaces on horses which carry the harness, on cattle the yoke, and the plantar cushion of a dog's paws—present sometimes circumscribed epidermic neoformations more or less elevated, with irregular edges, analogous in their pathogeny to the *corns* of men ; they are called *cores*. . . . Except in rare cases cores of our large animals are painless, and do not interfere with their use. It is not so with those of the paws of dogs ; they have deep roots which consume the papillæ," etc.

"Stickfasts are prevented by watching the condition of the harness and its paddings. As soon as the mortification sets in, its spreading is prevented by removing the pressure made on the spot ; the animal should, according to the case, be kept out of work for a certain length of time" (p. 226).

The various "modalities" of moist eczema are treated with dusting powders, but "the tegumentary vegetations must be amputated with the bistoury or scissors" (p. 229). We sometimes wish, in struggling through this extraordinary production, that we could amputate the Professor's narrative in the same way. After a great deal of research we find that "scratches" is the Professor's synonym for cracked heels, and that bruising of the skin of the withers is caused in the following way : "Let the harness fitting badly shave the skin, compress it, or bruise it, or let the tegument, moist with perspiration, adhere to the harness, or loosen with it, lacerating the subcutaneous connective tissue, then a phlemesia involving the skin and that tissue will develop, the region will become swollen, hot, and more or less painful" (p. 232). If that is not sufficient to make Reason totter on her throne, and, incidentally, to humble the pride of all Government-educated Bengali babus, the reviewer would like to know what is. And all this pother, if you please, about a simple harness gall !

Sebaceous cysts however reduce the Professor to absolute incoherence : "If the positive diagnosis in some cases is quite difficult, it is of secondary importance : the sebaceous cyst cannot be mistaken only for affections requiring the same mode of treatment—puncture or removal" (p. 241). We think a reward might fittingly be offered for any satisfactory explanation of the above.

In cutaneous horns, we are told, "the bacillus of Koch promotes the keratogenesis," a statement we would not, under any circumstances whatever, venture to call in question ; though we can scarcely extend the same tolerance to the following, which suggests a popular shibboleth : "The leg should be kept extended with an unremovable bandage in cases of a section of an extensor and flexed if there is section of a flexor : attempts must be made to bring both divided ends together by sutures ; but generally they are cut by the threads. Complications must be guarded against ; dressings must be applied on the legs ; on the trunk, ordinarily one is satisfied with washings and the use of absorbing powders" (p. 262).

In speaking of rupture of the coraco-radialis tendon the Professor says : "Professor Robertson had an animal destroyed because of severe osteoporosis. There was a great deformy, his body hanging between both fore legs, it, the scapulæ, having a horizontal position" (p. 264).

Professor Liautard is a noteworthy, if an unconscious humorist, and we thank him for having afforded us an hour or two of real enjoyment. It would not, however, be fair to extract any more of his *bons mots*.

A Text-book of Veterinary Medicine. Vol. IV. Infectious Diseases. By James Law, F.R.C.V.S. Ithaca : Published by the Author, 1902.

OF Professor Law's latest volume we can speak in terms of warm praise. Not only does it cover a wide field, but its information is accurate and recent. The work can fairly claim to represent the present state of clinical knowledge regarding infectious diseases of animals. The preparation of so important a volume must have involved an amount of reading and translation which few veterinary surgeons could accomplish, and the selection and collection of the vast amount of information here gathered together must have proved a task of great difficulty. For its successful accomplishment Professor Law, as Director of the New York State Veterinary College, is peculiarly well situated. but even with the co-operation of numerous disciplined assistants few writers could hope to better his performance. Professor Law has read, marked, learned, and inwardly digested most of the recent utterances of the best English, French, German, and Italian authorities. Adding to these the experience gained during a long period of practice and teaching in the United States, he has moulded the information into a harmonious whole. The plan of his work presents many analogies with the masterly *Equine Medicine* of the late Professor Robertson, upon which, however, has been grafted the information gained from the works of Friedberger and Fröhner, Nocard, Lignières and Cadéac, and a host of others. Reference to such subjects as glanders shows that Professor Law is in the unfortunate position of unsuccessfully urging upon an unwilling Legislature the need for a complete alteration in the laws relating to contagious diseases in animals. Although most of the States have passed acts requiring veterinary surgeons and owners to report the existence of glanders and other contagious diseases, yet they have omitted to give any practical incentives for such work, and it is quite evident from Professor Law's confession that the law is systematically evaded. Some years ago he endeavoured to have this condition of affairs remedied, and since that time he has used every effort to advance reforms.

In view of the sensation produced by Professor Koch's enunciation regarding the conveyance of tuberculosis from animals to men, it is interesting to peruse the many interesting and well authenticated cases brought forward by Professor Law to prove the direct communication of bovine tuberculosis to human beings. Not the least striking of these is afforded by the American Indians who receive from the Government a regular supply of meat. This meat is often diseased, and the internal organs are devoured raw, the flesh being eaten later as pounded preserved meat, still uncooked. The deaths of these Indians from tuberculosis is 50 per cent. of the total mortality. Dr Washington Matthews, who spent twenty-one years among the Indians, gives their food as the chief cause of the disease, and states that when the supply of fresh meat is liberal the death-rate is always highest.

A glance at the chapter on "Tetanus" shows that the trend of European work is closely followed in America, so that we find full and careful reference to the treatment by antitoxin and the still more recent treatment by brain emulsion. These references are characteristic of the whole work.

The book is necessarily too extended for the student, or even for the busy practitioner, but as a work of reference it will be highly valued, and in company with its three companion volumes will form a valuable addition to the literature of veterinary medicine.

Fleming's Text-book of Operative Veterinary Surgery. Volume I. Second Edition. Edited by J. Macqueen, F.R.C.V.S., Professor at the Royal Veterinary College, London. London: Bailliere, Tindall, & Cox, 1903.

WE recently had occasion to review the Second Volume of this work, which the late Dr Fleming left unfinished at the time of his death, but which was completed by Professor Owen Williams. It is fortunate that the publishers have now been able to issue the second edition of Volume I, which had necessarily become a little out of date. The fact that this edition has been revised by Professor Macqueen will be generally accepted as a guarantee that the work has been well done.

The book, which extends to 280 pages, is divided into four chapters, the first dealing with the manner of securing animals for operation, the second with the employment of anæsthetics, the third with elementary operative surgery, and the fourth (amounting to nearly half the volume) with general operations. A strong feature of the work is the profusion of illustrations, which number no fewer than 343, and are mostly excellent.

The book will be indispensable to English-speaking veterinary students, and it may be cordially recommended to practitioners who wish to keep themselves abreast of the times in matters relating to surgery.

The Pathology and Differential Diagnosis of Infectious Diseases of Animals. By Veranus Alva Moore, B.S., M.D., Professor of Comparative Pathology, Bacteriology, and Meat-Inspection, New York State Veterinary College, Cornell University. Ithaca, N.Y.: Taylor & Carpenter, 1902.

THIS work contains an Introduction from the pen of Dr Salmon, Chief of the Bureau of Animal Industry, United States Department of Agriculture, in which it is stated that an elementary treatise on the pathology of the infectious diseases of animals—a treatise that states briefly, clearly, and comprehensively all that is known and excludes all that is not known—has long been needed, and will be welcomed and appreciated. This is a somewhat dangerous remark to appear in an introduction, for it seems to claim for the work introduced a degree of excellence that borders on the unattainable. It is only right to add, however, that Dr Salmon was careful to explain that when he wrote this he had not seen the manuscript. We consider Dr Moore's book upon the whole a creditable one, but, alas, the perfect treatise on veterinary pathology—the one that will omit nothing that is known and include nothing that is uncertain—has yet to be written.

The opening chapter is devoted to a general consideration of etiology, infection, and specific infectious diseases. One might take exception to a good deal that is contained in this chapter, on the ground that no student will be able to understand or remember it unless he has previously made himself acquainted with the matters dealt with in the later part of the book. Some of the author's definitions and explanation of technical terms are not very happy, such as those of "wound infection," "septicaemia," and "specific infectious disease." The reader is told that if the invading organism happens to be that of a specific disease, giving rise to a definite series of symptoms and lesions, the affection is designated a specific infectious disease. This is the sort of statement that is likely to give a headache to the student of only moderate intelligence, but in the end he will probably conclude that the author wished him to understand that if a disease is specific it is also infectious. Needless to say, that is a wrong statement. Later on the author defines a specific infectious disease as the result of the multiplication within the animal body of a single species of micro-organisms! This might be correct

as a definition of what is meant by a "specific bacterial disease," but it is obvious that an essential quality of a "specific infectious disease" must be a tendency to spread by infection. The author is evidently prepared to maintain that every bacterial disease is an infectious disease, but in the sense in which the word infectious has hitherto been generally employed there are, fortunately, many bacterial diseases that are not infectious.

The author's attempts to make this question of specificity clear to the student are positively distressing. After giving the above-quoted definition of a specific infectious disease, he goes on to say: "The lesions may be localised or general, but the cause producing them is always the same. The bacterium anthracis will produce a disease which is called anthrax; no other cause can produce it, and no matter how much the lesions may differ in different individuals, if they are produced by this species of bacteria the disease is anthrax." Surely this would have been better expressed by saying that there is a micro-organism with certain well-defined characters which we agree to call the bacillus anthracis, and that we further agree to restrict the use of the term anthrax to the diseased processes for which this bacillus is responsible.

The second chapter is assigned to the diseases caused by streptococci, including, of course, strangles. It is curious to find a professor of veterinary pathology and bacteriology expressing himself in such an uncertain way with regard to the etiology of this disease. On this subject the author says: "Strangles is supposed to be caused by *Streptococcus equi*, first described by Schütz in 1888." The phraseology may indicate caution, but it is not likely to inspire the reader with confidence. Is strangles such a rare disease in the United States that the professor of pathology and bacteriology at one of the American veterinary colleges has not seen sufficient material to enable him to form a first-hand opinion as to the constancy of streptococci in the lesions?

Chapters III. and IV. are devoted to the remaining diseases caused by bacteria; while the morbid conditions caused by the higher fungi are discussed in Chapter V. The remaining three chapters are assigned respectively to the diseases caused by protozoa, to infectious diseases of uncertain etiology, and to a few diseases caused by animal parasites, while an appendix treats of disinfection. For the most part the author's descriptions afford evidence both of knowledge and care, but here and there one comes across a sentence which commits the double offence of making an erroneous statement and expressing it in a bad form. The following is an example. In referring to inoculation as a means of protecting cattle against contagious pleuro-pneumonia, it is said: "The specific nature of the tumor produced by inoculation is disputed, because an exactly similar tumor appears after the inoculation of pus or milk. We must also bear in mind that in those countries in which inoculation has been practised the disease shows no decrease, as, for instance, in England, where the official report lays particular stress on the doubtful value of obligatory inoculation for pleuro-pneumonia as in France and Belgium where inoculation was obligatory." The author appears to be under the erroneous impression that inoculation was much practised in England, and that the disease shows no decrease, whereas the facts are that only a small minority of outbreaks in England were ever treated by inoculation, and the disease is now non-existent in this country. At this date it is remarkable to find a bacteriologist who seriously believes that the peculiar inflammatory effects which follow inoculation with pleuro-pneumonia lymph can be produced by the injection of milk.

There is one other feature of the work that is open to adverse criticism, namely the relative amount of space that has been assigned to the description of the different diseases. Strangles, which is a very important disease, at least in Europe, is dealt with in four pages, while nearly two and a half

pages are assigned to "apoplectiform septicæmia in chickens." Rather less than nine pages is allowed for black-quarter (which, by the way, the author terms "symptomatic anthrax"), while twelve are required for the description of "infectious entero-hepatitis in turkeys."

There are 7 Plates and 73 smaller illustrations in the text, and the majority of these are good.

Methods and Theory of Physiological Histology. By Gustav Mann. M.D., C.M., Edin., B.Sc. Oxon., Senior Demonstrator of Physiology in the University of Oxford. Oxford: Clarendon Press, 1902.

IN the world of physiology and pathology the name of Dr Gustav Mann has for a good many years been well known as that of a laborious worker in connection with micro-anatomy and micro-physiology, and there can be no doubt that the present work will add greatly to the reputation that he had already gained. It is not, like some other books with a similar title, a mere compendium of formulæ and staining prescriptions, but a philosophical treatise in which an earnest attempt is made to unravel and explain the physics and the chemistry of the methods employed in the study of the ultimate structure and composition of living cells and their derivatives. Naturally, among these methods fixation and staining come in for a very large share of attention, but such subjects as bleaching, isolating, decalcifying, injecting blood and lymph vessels, methods of obtaining sections, and microtomes, are also fully dealt with. Although we have said that the work is no mere collection of formulæ, it contains a description of all the methods of staining, fixing, etc., which have hitherto been published, accompanied by much valuable advice as to the selection of methods and reagents for particular purposes in histological work. The work, in short, is one that everyone engaged in histological work, whether normal or pathological, will be glad to have constantly at his hand.

Leitfaden für Fleischbeschauer. Von Dr R. Ostertag, Professor an der Tierärztlichen Hochschule zu Berlin. Berlin: Richard Schoetz, 1903.

Wandtafeln für Fleischbeschau. By the same Author and Publisher.

THE first of these is a small handbook of 205 pages, designed to meet the requirements of the lay or non-veterinary meat inspectors, who, under the laws relating to the subject, have a definite and restricted rôle assigned to them in Germany. Such lay inspectors have to pass an examination before a Board of veterinary surgeons, and in order to qualify themselves for that examination they have to attend a public slaughter-house for not less than four weeks, and during that period receive theoretical and practical instruction in the inspection of meat.

Licensed lay inspectors have their duties limited as follows: Inspection before slaughter can only be carried out by them on:—

1. Perfectly healthy cattle, calves, sheep, goats, swine, or dogs.
2. Animals which exhibit only the symptoms of unimportant diseases (without serious constitutional disturbance).
3. Animals which not more than twelve hours previously have experienced a fracture of a bone, or other similar injury.

After slaughter they can only examine or give an opinion regarding:—

1. Perfectly healthy animals.
2. Slight and easily recognisable abnormalities.
3. Carcases or organs surrendered by the owner as unfit for food.

With this explanation of what is expected of the lay meat inspector, we need only add that Professor Ostertag's latest work is certain to prove of great value to the class for which it is intended. It contains a large number of excellent illustrations.

The wall-diagrams by the same author are six in number. The first contains figures of the ox, calf, sheep, and pig, with lines indicating the customary methods of dividing the carcass; the second is devoted to the teeth of the ox, sheep, and pig, as indicative of age; and the remaining four are intended to serve as guides to the position of the various groups of lymphatic glands in the same animals. The cost of the series is 20 mark (£1). Both teachers and students will find them useful.

CLINICAL ARTICLES.

OBSTRUCTION OF THE COLON DUE TO A CALCULUS.

By G. P. MALE, M.R.C.V.S., House Surgeon, Royal Veterinary College, London.

AN aged cab mare was admitted to the infirmary at 7 A.M., 2nd March, showing symptoms of abdominal pain.

For two days previously no fæces had been passed, and on the preceding evening indications of pain had first been noticed.

There was no history of a previous attack.

After admission she showed dull pain, lying down at intervals; pulse, 60 and full; temperature, 101° F.; respirations, 16; in fact, all the symptoms of impaction of the colon.

On exploration, the rectum was found to be dry and ballooned, and an accumulation of fæces was found in the double colon.

A ball containing carbonate of ammonia and nux vomica was administered, followed by a hypodermic injection of eserine sulphate 1½ grs., and pilocarpine 1½ grs., and frequent enemata.

At 3 P.M. a drench containing 2 ounces of oil of turpentine and 1½ ounces of aromatic spirits of ammonia in a pint of linseed oil was administered.

At 9 P.M., the mare being still in pain, a similar injection of eserine and pilocarpine was given to excite peristalsis.

3rd March. Pain still manifested by turning the head to the flank and lying down. The temperature was now 103° F., respirations, 30, and the mucous membranes much injected.

Eserine and pilocarpine 1½ grs. were again administered, but, having no effect, a 3iv. physic ball was given.

4th March. Still no action of the bowels. The mare was now weaker; the temperature 103° F., pulse 80, fluttering and irregular.

Ammonium carbonate 3ij. and ginger 3j. were exhibited at intervals during the whole of the three days.

Towards evening a small quantity of liquid fæces were voided, the mare straining somewhat.

5th, 6th, and 7th March. Pain was absent, the bowels acted

freely and at frequent intervals, and towards evening of the 7th chlorodyne $\frac{3}{4}$ j. was given.

8th, 9th, and 10th March. Mare looking brighter, feeding a little, and fæces of normal consistence.

11th March. Some indications of pain apparent and off food.

12th March. About midday she again showed pain, with symptoms similar to those of the 2nd inst.

On exploring per rectum the bowels appeared empty as far as the hand could reach.

13th March. The mare was easier and ate a small quantity of food. Towards evening, however, pain was again shown, and this continued, with intervals of ease varying from about 6-15 hours, throughout the next two days. During the whole of this time, *i.e.*, from the 10th to 16th, the action of the bowels continued in abeyance.

Chloral hydrate in $\frac{3}{4}$ j. doses was given to relieve the pain.

16th March. To facilitate examination the mare was cast with hobbles, and on exploration per rectum a hard body could be felt with the tips of the fingers, though it was difficult to say whether it was in the large or small colon.

The idea suggested itself that here was an opportunity to attempt the operation of laparotomy.

The owner's consent having been obtained, this was accordingly done on the 17th.

Following the lines laid down by Professor Macqueen in his experimental cases in 1895, the flank was opened on the right side for about eight to ten inches downwards and forwards from a point midway between the angle of the haunch and the last rib. Previous to the operation the horse was cast and chloroformed, the flank carefully shaved and disinfected, and cloths soaked in disinfectant spread on each side of the operation area. Three incisions were made, one through the skin and external oblique muscle, after which the top leg was stretched in a backward direction and fastened there; the second through the internal oblique muscle; and the third through the transversalis; the two latter incisions being in the direction of the muscular fibres, to act as a valve when sutured again. The hæmorrhage having been stopped, the peritoneum was ruptured, the arm introduced, and a large calculus was found far forwards, apparently in the fourth part of the double colon.

This bowel being fixed to the first part of the colon, to the cæcum, and to the abdominal walls, I found it quite impossible without rupturing the bowel walls to bring it within one foot of the flank incision. The muscles were accordingly sutured in layers with thick cat-gut, and the skin brought together with No. 6 Chinese twist, and the mare allowed to rise.

The same evening, for humane and economic reasons, the mare was slaughtered.

A *post-mortem* examination being made, a calculus was found at the end of the double colon, just before it narrows down to form the small colon. The bowels were inflamed at this point, though not very markedly, and contained soft fæces. The calculus was oval in shape, with a corrugated surface; it measured 21 inches in its larger and $16\frac{1}{2}$ inches in its smaller circumference, and weighed $3\frac{3}{4}$ lbs. It appeared to be composed of compacted dust particles.

The above case shows that although laparo-enterotomy may be successfully performed when the calculus is situated in the small colon, yet it is quite impracticable when the obstruction is in either the fourth or first part of the double colon. It is therefore of great moment to carefully ascertain the exact position of the calculus before advising operation.

GASTROTOMY IN A DOG.

By PETER WILSON, M.R.C.V.S., Lanark.

Subject.—A three-months-old prize-bred fox-terrier puppy.

History.—Early in October 1902 the owner spoke to me about it picking up pieces of iron in his forge, and said he thought it had swallowed some of these. I advised him to give it an emetic, to see if it would vomit any. He did so, but with no result. At this time it was eating well.

However, by Saturday, the 25th October, its appetite was so bad that it could scarcely be induced to touch any food, and, on lifting and shaking it gently, the owner heard a sound like that produced by shaking a bag of nails. He brought it in to me that night.

Symptoms.—Its general appearance was unthrifty, and it was very thin, but fairly lively.

The previous history led me to examine the region of the stomach. Here one could feel, on palpation, something hard; on auscultation, a sound such as might be produced by a miniature stone-crusher was heard; and, when shaken, a sound of iron rattling was distinctly audible. There being no doubt about the diagnosis, I informed the owner that the only chance of saving the animal's life lay in an operation, and we arranged to perform it next day.

Operation.—In the morning I washed him in lysol solution.

When the owner arrived, we shaved the seat of operation, and washed it with chinosol solution, then gave an anæsthetic (æther was principally used, with a little chloroform), scrubbed the part with æther, and again washed with chinosol. The operation was performed according to the directions in Professor Hobday's "Canine Surgery," only in suturing the abdominal wound the peritoneum and muscles were sutured together. Silk was the suture material used for this and the stomach, and aseptic gut was used for the skin. To finish with, a stripe of sublimate wool was placed round the abdomen, and fixed with a bandage. The wound gave no trouble. It healed by primary intention.

After Treatment.—The patient was kept for two days and a half on enemata of extract of beef, getting water with a little boric acid to drink. From then till the ninth day it got milk and water and beef tea to drink. Afterwards it got porridge, bread with milk, and raw minced beef. It went home in fourteen days in good health, and has remained so.

Result of Operation.—There were removed from the stomach fifty-eight pieces of iron, weighing rather more than a quarter of a pound. They were mostly old shoe nails, with a few old rivets, and several splinters of iron.

ANGIOMA OF THE SKIN OF A COW.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

SOME time ago my attention was directed to a pedunculated growth of the skin covering the right mamma of an aged Ayrshire cow. It was situated on the posterior aspect, several inches below the junction of the mamma with the thigh. It was the size of a pigeon's egg. The skin which covered it was thin, of a bluish colour, and showed comparatively few hairs. The tumour was painless, soft, non-pulsating, and it bled profusely on being roughly handled. The blood, as it escaped, flowed over the skin of the mamma, and dropped on the ground, or, if milking operations were being performed, in the vicinity of, or into the milk pail. The tumour was removed by *écraseur*.

No reliable clinical history was obtainable.

On microscopical examination, the tumour was found to be composed of a more or less delicate network of fibrous tissue. The spaces formed by this network varied greatly in size. They were, as a rule, circular, oblong, or elongated according to the angle at which the vessel was cut. Some, however, were irregular in shape owing to the trabeculæ becoming ruptured. These spaces, which were filled with blood, and lined by flattened, spindle-shaped, endothelial cells possessing one or more round nuclei, were apparently merely dilated blood vessels. The trabeculæ were in some places composed only of the two layers of endothelial cells lining the respective spaces, but more frequently these cells were supported by an intermediate layer of fibrous tissue. Numerous young connective tissue corpuscles were present between or in the fibres composing the trabeculæ. The tissues in the vicinity of the base of the tumour showed a dense cellular infiltration. This was evidently due to some inflammatory process — a complication which occasionally occurs in these tumours.

DERMOID CYST IN A COW.

By the Same.

WHILST making a *post-mortem* examination on an aged Irish cow, a growth was discovered on manipulation under the mastoido-humeralis, in the lower part of the cervical region. On dissection, it was found to be situated in close contiguity, but inferior to the right prescapular group of lymphatic glands. It was loosely attached by fibrous tissue to the inner surface of the mastoido-humeralis and to the adjacent adipose tissue. It was irregularly circular in shape, and had the appearance of a sphere flattened laterally through pressure. It measured 6·4 cm. in diameter, and 3 cm. in thickness. It was firm and non-elastic in consistency.

The growth on section was seen to be a dermoid cyst.

The cyst wall measured 1 mm. in thickness, and appeared on macroscopical examination to be structureless. The inner surface was white, and sparsely studded with hairs. The latter were white, and finer than those usually found on cattle. In some instances they attained two inches in length. The contents of the cyst were shed dermoid

hairs, matted together and mixed with a yellow inodourless material, evidently the secretion of sebaceous glands.

This material, on microscopical examination, was found to be largely composed of granular detritus. The cyst wall showed the typical formation of skin. It was sharply divided into derma and epidermis. The papillæ were small in size and few in number. The derma was composed of fibrous tissue, in which non-striated muscle



Section Showing Structure of Cyst-Wall ($\times 65$).
A, Epidermis; B, Derma; C, Hair Follicle.

fibres were present. In this layer were hair follicles with sebaceous glands, but no evidence of the existence of sudoriparous glands could be determined. The epidermis varied in thickness, and in places it showed to perfection the different strata. The germinal layer was composed of serrated columnar cells. The superincumbent cells were polygonal in shape, but towards the surface they became more and more flattened, and eventually their nuclei disappeared. The stratum lucidum was in places well marked. The stratum corneum frequently exceeded in thickness the stratum lucidum and stratum granulosum.

CLINICAL NOTES.

By WILLIAM ROBB, F.R.C.V.S., Glasgow.

COCAINE.

ABOUT a year ago, in this Journal, I described an experience I had had in performing plantar neurectomy under cocaine. Since that I have repeated it with similar results. The subjects were a half-bred hunter, on both fore legs of which I intended operating, and a nearly thoroughbred saddle-hack, of which one fore leg was operated on. I commenced with the big horse first, and got one leg done without any trouble. I then took the blood mare, and after a little trouble I got the needle inserted, and operated without further evidence of pain. This left me with still one leg of the half-bred to

do, and, as before, he stood perfectly quiet when the needle was passed through the skin. I waited fully ten minutes; but this time he would not stand the incision. I waited some time longer, tried again with same result, and, thinking that I might have injected into the blood-vessel, I tried the other side, but never got further than the incision. I then injected more cocaine, but had to give it up and cast the animal to get the operation finished.

This almost coincides with my previous experience, as it was the second leg that I had trouble with on that occasion. From my experience I believe cocaine in large doses acts as an excitant, but why it should fail to produce local anæsthesia, as in the above case, I am unable to say. Since that last experience I have discarded cocaine for chloroform, even when cast, as I find it more satisfactory when operating, and I do not have the thickening of the leg that sometimes follows cocaine injections. As to the time that should be allowed to elapse between injections and commencing to operate, I have never been quite certain, but have always tried it between ten and twenty minutes after injection. A friend of mine, Mr Alex. Milligan, M.R.C.V.S., Paris, who, I believe, has an almost unique experience in the use of cocaine, informs me that he never starts until he has allowed at least a period of twenty-five minutes to elapse, and generally allows thirty-five.

AMPUTATION OF PENIS.

Within the last three months I have had this operation to perform twice, and in both cases I operated by ligaturing the stump. In a previous case I operated by ligaturing the blood-vessels, but from the experience gained in the last two I believe the former method the easier, and the results quite as satisfactory. I admit I liked the appearance of the stump much better in the case in which I ligatured each vessel independently, but I confess to a more comfortable feeling after the operation was over in my last two cases.

The first of my recent cases was operated on on account of a large tumour affecting the glans. After I had dissected out the urethra, I passed the chain of the écraseur round the stump and screwed up until the chain broke. I then simply ligatured the whole stump behind the chain, and, after cutting it off, stitched back the urethra in the usual manner.

My last case was that of a six-years old Clydesdale stallion, who had only partial control of his penis, the result being that about a third of it protruded out of the sheath. The question put to me by the owner was this: "If you remove the portion that is always hanging out, will he be fit to serve mares?" To help me to make up my mind, I had him tried with a mare, so that I might observe the kind of erection that took place.

The result of this was a decided improvement in the condition of that organ, but it lacked the normal rigidity and elevating power of a sound penis. I then decided to remove sufficient, so that in ordinary conditions it would be kept within the sheath. There was a point that cropped up in my mind at that time, as to the part played by the glans in the act of coition. Does the removal of the glans, in a penis that can become erect and eject semen, destroy the functional

powers of the organ? My previous patients being geldings, I was prepared for a better development of the structures in the stallion. At the same time I was a little taken aback at the amount of fascia and the quantity of muscular fibre I had to remove before exposing the urethra.

In this case I simply screwed up the *écraseur* as far as I judged safe without breaking the chain. I then passed a needle with a stout silk ligature attached through the stump, and left the ends untied. I repeated this again, finally tying the inferior one above and the superior one below. Having cut off the portion anterior to the ligature, I split the urethra and stitched it back.

From my experience, I would strongly advise anyone operating always to dissect out at least two inches of urethra. A certain amount of contraction takes place at the time of operation, and also afterwards in the healing process, and when one has plenty of urethra to work on it makes the stitching easier, and I believe tends to prevent constriction afterwards. Any excess of urethra is easily removed by trimming with scissors.

ABSCESS IN LEVATOR HUMERI.

In my experience this affection, with one exception, has been confined to heavy draught horses, the exception being a vanner. I have never seen it in driving or saddle horses. The abscess is, in recently affected cases, usually found in the upper third of the tumour, and there is not much alteration in the structure of the muscle. Even if the animal is idle and under such treatment as fomentation, blistering, or setoning, considerable fibroid change takes place in the belly of the muscle, and the cavity containing the pus gets smaller. I have tried these methods, and they seldom make any difference in this class of shoulder-tumour. Even when absorption did take place, on the animal being sent to work the tumour returned. There is but one treatment that will give general satisfaction, viz., opening into the abscess in the centre of the swelling. At one time I also removed a considerable portion of the fibrous growth, but do not now believe that this is necessary. Later on I contented myself with removing a wedge-shaped portion out of the centre of the growth. More recently I have, when operating, simply made an incision extending the full length of the fibrous growth and deep into the pocket containing the pus. There is considerable bleeding, but it is easily controlled by plugging and stitching the skin wound. The following day remove the bottom stitch, take out the plug, wash out the wound, and re-plug.

There is one point that I have noted as necessary—it is to see that the external (skin) wound is longer than the internal wound. If it is not, there is tendency for the escaping discharge to lodge behind the skin and burrow downwards.

I have had quite a number of this type of case recently, and found on an average they were fit for work in about a month.

Abstracts and Reports.

THE HISTOLOGICAL DIAGNOSIS OF RABIES IN THE DOG.

It is well-known that the diagnosis of rabies in the dog by mere *post-mortem* inspection of the organs presents serious difficulties. On the other hand, the method of diagnosis by experimental inoculation of the rabbit is open to the serious objection that it demands a period of not less than ten or twelve days, and more frequently requires fifteen days.

Between the years 1886 and 1892 Babes insisted on the presence of distinct alterations in the nerve cells of the medulla oblongata and spinal cord of rabid dogs, and on the possibility of basing a diagnosis on the presence or absence of such lesions. According to that author, if one found in the grey matter nodules composed of embryonic tissue, accompanied by a perivascular growth of the same kind, and breaking up of the chromatin granules, one might affirm with certainty that the dog had been the subject of rabies.

The statements by Babes did not receive much attention until two years ago, when two Belgian histologists, Van Gehuchten and Nélis, pointed out the existence in the nervous system of animals dead of rabies, and especially in the dog, of characteristic lesions, the *post-mortem* recognition of which enables one to make a rapid and precise diagnosis.

According to these authors, the most marked and constant lesions, and also those which set in soonest, have their seat in the peripheral ganglia of the cerebro-spinal and sympathetic systems. In the normal condition these ganglia, when examined microscopically, are found to be composed of a supporting tissue with capsules, each of which encloses a single nerve cell. The lesions characteristic of rabies consist in atrophy, invasion, and destruction of these nerve cells by newly formed cells, which appear between them and their endothelial capsules. At the outset the nerve cells are first compressed by swelling of the endothelial cells of the capsule, and then by the newly formed cells interposed between the capsule and the nerve cell. Finally the fixed cells of the part invade the nerve cell, and determine its complete destruction by occupying the whole cavity of the endothelial capsule. The little colonies of cells thus produced form the miliary granulations first pointed out and described by Babes under the name of "rabid tubercles."

These lesions of the cerebro-spinal ganglia have been observed in the dog, cat, rabbit, and human subject. Nélis and Van Gehuchten assert that they are characteristic, and constantly present in individuals who have succumbed to natural rabies, although some variations are observable in the lesions according to the particular ganglia, the animal species in which they occur, and also the individual.

The lesions are much more pronounced in the dog than in the rabbit. The cerebral ganglia always show the most marked lesions, notably the ganglion of the pneumogastric nerve. It is this ganglion which is most conveniently submitted to examination with a view to rapid diagnosis. The observations of Babes and Van Gehuchten have been confirmed by a considerable number of observers, including Rabieaux.

Rabieaux examined 32 cases of rabies in the dog, 2 in cats, 1 in a donkey, and 2 in goats; and in every case he was able to detect the ganglionic lesions described by Nélis and Van Gehuchten. The lesions varied somewhat in their distinctness, but were never entirely absent.

Rabieaux found that in dogs killed during the period of evolution of the disease the lesions are pretty frequently absent (11 times out of 29). The

rapidity with which the lesions appear is very variable. Sometimes they were observable in dogs killed immediately after the onset of the symptoms, while they were absent in others destroyed at a later period of the disease, when the furious symptoms had set in.

The observations were controlled by the examination of ganglia taken from seventeen dogs dead of other diseases than rabies, and in none of these cases were analogous lesions present.

The experience of Rabieaux and of all the other authors who have investigated the subject shows that the practical value of this method of diagnosis varies according to the nature of the case. If the suspected animal has been killed at an early stage of the disease its value is very restricted, for the lesions are then at the most very slight, and most frequently entirely absent. Consequently, the non-detection of the lesions never justifies one in pronouncing the case not rabies. On account of its uncertainty, the method is therefore practically useless in such circumstances.

On the other hand, in the case of animals that have died the before-described lesions are constantly present, and such lesions have not hitherto been observed with certainty in animals dead from other diseases than rabies.—(*Journal de Méd. Vét.*, December 1902.)

THE SERO-DIAGNOSIS OF GLANDERS.

In the "*Journal de Médecine Vétérinaire*" (August 1902), M. Rabieaux records the results of his observations with regard to the value of sero-diagnosis in suspected cases of glanders. These observations were made on serum taken from nine glandered horses (one case being acute and the others chronic), and from eleven healthy horses either under treatment in the hospital or purchased for the purpose of dissection or operation.

His method of procedure is as follows:—

The serum to be examined is collected as pure as possible, and diluted with sterile distilled water in proportions varying from 1 in 10 to 1 in 1500. The diluted serum is then mixed in small sterile tubes with an equal volume of culture of the glanders bacillus in peptonised bouillon (without glycerine), the age of the cultures being from 24 to 72 hours. The mixture thus obtained is placed in the incubator at a temperature of 35° to 37°, and the tubes are examined at variable intervals under the microscope. In every case control observations were made by preparing in an identical way mixtures containing serum from a horse known to be not glandered.

Rabieaux found that at the temperature mentioned both glandered and non-glandered serum possessed the power of agglutination, but there was a marked difference between the two in respect of the rapidity and intensity of the agglutination, the intensity being measured by the degree of dilution with which the phenomenon could still be observed.

With dilutions of from 1 in 10 to 1 in 50, the difference between the agglutinating power of the two kinds of serum was not very manifest. As a rule, however, in dilutions of the same strength the glandered serum more rapidly produced compact masses, with very few free mobile bacilli between them. Whereas with glandered serum, the phenomenon is observable in from 20 minutes to 3 hours, from 2 to 6 hours are required for its production with non-glandered serum.

With dilutions of from 1 in 100 to 1 in 250 the difference between the activity of the two kinds of serum is a little more marked. In from 8 to 18 hours the non-glandered serum determines the agglutination of the bacilli in masses, whereas the same effect is produced with glandered serum in from 1

to 10 hours. Moreover, with such dilutions it not rarely happens that the non-glandered serum does not agglutinate at all. Rabieaux, therefore, considers that reliance cannot be placed upon the results obtained unless the serum is employed in mixtures considerably more diluted. He found that when the dilution exceeded 1 in 300 or 1 in 400 (11 observations), the non-glandered serum never agglutinated, whereas if the serum came from a glandered animal agglutination could be obtained with dilutions of from 1 in 500 to 1 in 1000, or even 1 in 1500.

In view of his own results and those obtained by other observers, he thinks that whenever a serum exhibits agglutinating property in the dilution of 1 in 1000 the animal from which the serum was obtained ought to be considered glandered.

All the experimental conditions remaining the same, it was found that the rapidity with which the agglutination was produced varied a great deal according to the individual, and this applied both to the glandered and non-glandered subjects. Thus, in two observations a very obviously positive result was obtained in from 2 to 3 hours with dilutions of 1 in 1000, although, as a rule, with such a dilution the phenomenon does not become distinct until between the eighteenth and thirty-sixth hour. Very exceptionally it required 48 hours, but the interval never exceeded 72 hours. No explanation can yet be offered of these individual peculiarities, but the interesting fact was observed that the agglutination manifested itself in a very intense fashion (2 to 6 hours with dilution of 1 in 1500) when the serum was furnished by a glandered subject with an elevated temperature. This fact is of practical importance, since the mallein test cannot be carried out on such subjects.

The aspect of the phenomenon under the microscope varies a little in different cases. Sometimes almost the whole of the bacilli become collected together in large compact clumps, very few of the organisms remaining free. At other times there are numerous free bacilli between clumps composed of from 15 to 50 more or less loosely united bacilli. In dealing with serum having high agglutinating power, especially when much diluted, it was observed that the bacilli, in multiplying, frequently remained collected together so as to form short chains of three or four.

The phenomenon of agglutination may sometimes be made visible to the naked eye. With dilutions of 1 in 10 to 1 in 100, or exceptionally even 1 in 1000, the bacilli are observed to become united in small clumps, which fall to the bottom of the tube, leaving the liquid clear. When the tube is agitated the little whitish clumps do not produce a uniform turbidity, such as is seen in control cultures in the same conditions. This alteration, however, is not permanent, especially with feeble dilutions, for the agglutinated bacteria still possess their power of growth and multiplication, in consequence of which the liquid again becomes turbid.

The injection of mallein does not modify the agglutinating power of glandered serum, provided the serum is taken when the temperature has returned to the normal. The agglutinating power of the serum, however, appears to be increased if it is taken during the febrile period.

Observations were also made by Rabieaux with regard to the agglutinating effect at different temperatures. At the temperature of the laboratory, which fluctuated between 12° and 15° C., it was observed that the phenomenon of agglutination set in less rapidly, whether the serum was glandered or non-glandered, and whatever was the degree of dilution. This retardation of the agglutination was readily observable with strong dilutions, and it was especially manifest and well marked with extreme dilutions of either glandered or non-glandered serum.

At low temperatures (—3° to 5°) the phenomenon of agglutination is modified both with regard to the rapidity of its manifestation and its intensity. The clumps are later in making their appearance, and with extreme dilu-

tions they may be entirely absent although formed in the same dilutions at 12° to 15°, and still better at 37°. At these low temperatures the non-glandered serum almost constantly, although not in every case, lost its agglutinating power in dilutions of 1 in 100, and even glandered serum no longer agglutinates when diluted to the extent of 1 in 1000. The low temperature does not destroy the agglutinating power of the serum, for in mixtures in which it does not appear with 48 hours' exposure to such temperatures it may rapidly set in when they are transferred to the incubator at 35° to 37°.

M. Rabieaux draws the following conclusions from his observations:—

The very marked difference which exists between the agglutinating power of a serum according as it comes from a glandered or a non-glandered horse may be used as the basis of a method of diagnosing glanders.

Whenever a serum manifests the agglutinating property in the dilution of 1 in 1000, the animal which furnished it ought to be considered glandered.

The sero-diagnosis of glanders, in view of its technical character, must always remain a laboratory method. Consequently, it can neither supplant nor replace the experimental methods of diagnosis which are in common use, these being much more practicable for every one.

Nevertheless, the method of sero-diagnosis may find useful application in practice, either in order to corroborate other methods when the results furnished by these are indecisive, or to take the place of these when, for any reason, they cannot be carried out. Especially in cases when the animal is already dead, the method of sero-diagnosis may be usefully employed.

In carrying out the test, observations should always be made at the same time in precisely similar conditions with serum coming from a healthy subject.

It is advantageous to operate at a temperature of from 35° to 37°, but if the serum to be examined is not pure it is necessary to submit the mixture of serum and culture to a temperature of 60° or 65°, or to sterilise the serum previously, so as to prevent the accidentally present organisms from multiplying in the liquid.

TWO NEW TRYPANOSOMATA OF THE OX.

In a note communicated to the French Academy in the month of March, 1902, Laveran described under the name *trypanosoma theileri* a trypanosome discovered by Mr Theiler, veterinary surgeon, at Pretoria, in cattle coming from different parts of the Transvaal. The disease caused by this trypanosome is very widely spread throughout the whole of South Africa, where it is known under various names, the commonest being *galziekté* (gall sickness). The *tr. theileri* varies from 30 to 65 μ in length and from 2 to 4 μ in breadth.

Although it does not appear to be certain that ticks play any rôle in the propagation of gall-sickness, Theiler observed that these are specially numerous on the diseased animals. The ticks in question belonged to the *rhhipicephalus decoloratus* of Koch.

In the month of August last Theiler sent from Pretoria to Laveran some blood preparations from an ox. These showed a considerable number of trypanosomes belonging to a species different from the *tr. theileri*. Laveran gives to this new trypanosome the name of *tr. transvaaliense*. It varies somewhat in size, but is rather smaller than the *tr. theileri*. It measures from 18 to 50 μ long by 4 to 6 μ broad. The position of the centrosome with regard to the nucleus is characteristic. In all the trypanosomes hitherto known the centrosome is situated remote from the nucleus, generally a little distance from the posterior end. In the *tr. transvaaliense* the centrosome is always near the nucleus, often in contact with it, a fact which bears out the view

previously expressed by Laveran and Mesnil with regard to the nature of this chromatic body. Owing to the proximity of the nucleus to the centrosome, the undulating membrane is much less developed than in the *tr. theileri*.—(*Revue Vétérinaire*, 15th January 1903).

MAL DE CADERAS.

The principal diseases caused by trypanosomata are, surra in India, nagana in Africa, and mal de caderas in South America. These diseases and the parasites which cause them resemble one another so closely that one might be tempted to ask whether the three names do not cover a single disease, or, at the most, simple varieties of one disease.

MM. Laveran and Mesnil were fortunate enough to obtain for the purposes of study in the living condition both the trypanosoma of nagana (*tr. brucei*) and that of mal de caderas (*tr. equinum*), and as a result of their investigations they conclude that nagana and mal de caderas are two perfectly distinct diseases. The same animal species are susceptible to the *tr. brucei* and the *tr. equinum*, and the same species are refractory to these parasites. Mal de caderas is a disease which develops more slowly than nagana in some animal species, notably in the guinea-pig, and in some cases in the dog. As a rule paralysis of the hind quarters is more marked in the horse in mal de caderas than in nagana. On the other hand, hæmoglobinuria is common in mal de caderas and very rare in nagana.

Alongside of those differences there are many resemblances. For example, arsenious acid and human serum exercise the same action on both trypanosomata.

The differences between nagana and mal de caderas, on which is founded the conclusion that nagana and mal de caderas are distinct maladies, are the following :—

(1) There is a constant morphological difference between the parasites. This difference is in respect of the size of the centrosome, which is larger and stains more deeply and more readily in the case of *tr. brucei* than in *tr. equinum*.

(2) Animals immunised against nagana are still susceptible to mal de caderas.

(3) The serum of animals immunised against nagana, while effective against the *tr. brucei*, is without effect on the *tr. equinum*.—(*Ibid.*)

A CASE OF STRIATED MYOMA IN THE HORSE.

M. Monod, a French army veterinary surgeon, claims to have met with an example of this extremely rare tumour. An artillery mare in the month of February 1902 developed suddenly in the region of the shoulder a firm tumour which was well-defined, movable under the skin, but slightly adherent to the deeper tissues. The animal's general condition was not affected, and there was no lameness or impediment in the action of the limb.

The tumour was treated by massage, and under this it became somewhat smaller and softer. However, the improvement was only temporary, and the tumour was excised on the 12th March.

It was found to be easily enucleated, as it was encapsuled and contained in a pouch where it floated freely in a small quantity of limpid liquid. At its deeper part it was furnished with a long pedicle, which penetrated between the anterior border of the supraspinatus and the mastoido-humeralis and

turned round the anterior edge of the scapula, at which point it was cut off in order to free the tumour.

The wound cicatrised very rapidly. The tumour was completely surrounded by a very resistant fibrous envelope, and this was reflected on to the pedicle, with which it became continuous.

The whole tumour had the form of a mushroom, about 6 centimetres long by 5 broad and 5 thick. The convexity of the tumour was directed towards the skin. When incised the cut surface of the tumour had a uniform reddish-yellow colour, and the tissue of it was disposed in laminated strata running from the pedicle to the periphery. The pedicle itself appeared to be composed of fibrous tissue.

Microscopic examination of the tumour, made by Professor Cavalie of the Medical Faculty of Bordeaux, showed that it was composed almost exclusively of striped muscular tissue, arranged in bundles separated by connective tissue. The muscular fibres were striated, some of them being larger than the others. They were provided with sarcolemma and showed several peripheral nuclei. The muscular fibres terminated in conical points. The fibrils had the characters of those of normal striated muscular fibres.—(*Recueil de Méd. Vét.*, 30th June 1902).

GENERALISATION OF CARCINOMA OF THE MAMMARY GLAND IN THE BITCH AND CAT.

M. Pettit has placed on record two cases of primary carcinoma of the mammary gland—one in a bitch and the other in a cat—in which the disease became generalised.

In the case of the bitch the mammary glands contained numerous nodulated firm tumours, varying in size from a nut to an apple. The skin covering the tumours was intact but adherent. When scraped, the tissue of the tumours yielded very little juice. One of the tumours situated in the pectoral region had been almost entirely transformed into a cyst. There also existed immediately behind the right elbow a subcutaneous tumour as large as one's fist. This was white, soft, and easily disintegrated.

Microscopic examination showed that the case was one of primary carcinoma of the mammary gland, developed at the expense of the glandular epithelium. An interesting point in the case was that, together with the purely carcinomatous growths and closely resembling them in naked-eye appearance, there existed some typical fibro-adenomata, which are very generally and rightly regarded as benign tumours.

Secondary tumours histologically identical with the mammary carcinomata were present in the liver, spleen, and lungs. The tumours in the liver and lungs were very numerous, and varied in size from a pea to a nut. They might have been confounded with tubercles. One of the bronchial glands was notably enlarged, and destroyed in its central part, where it presented a cavity of a cancerous nature. The spleen contained three or four tumours towards its thick end. One of these was of the volume of a nut, and projected partly from the surface of the organ. The others were much smaller.

In the case of the cat, the mammary region was completely invaded by a tumour which microscopic examination proved to have the same histological structure as that of the bitch. The tumour had ulcerated, and presented a crater-like cavity on its surface, indicating its malignant nature.

In this case the cancer was generalised to the sub-lumbar lymphatic glands, and the abdominal wall, especially behind the sterum, was much thickened and infiltrated with nodules. The peritoneum was intact, but the kidneys contained a considerable number of tumours, varying in size and situated in

the cortical substance. These were visible through the capsule, and bore a close resemblance to tubercles. The spleen also contained a tumour of considerable size.

Extensive lesions were present in the thoracic cavity. The costal pleura was covered with tumours, some being as large as a pea, but most of them smaller. These were round and projecting, and could easily have been mistaken for tubercles. More numerous tumours of the same sort were present on the upper face of the sternum, and the prepectoral lymphatic glands were enlarged and invaded.

The lungs were firm and inelastic, and the lung tissue at first sight appeared to be diffusely infiltrated with a carcinomatous growth. Closer inspection showed that the new tissue was composed of very small greyish-white opaque growths. These were non-caseous, but in spite of this they might without histological examination have led one to suspect tuberculosis rather than carcinoma.—(*Ibid.*)

THE NON-IDENTITY OF HUMAN AND AVIAN DIPHTHERIA.

M. GUERIN has carried out an investigation bearing on the etiology of avian diphtheria, and has thereby obtained further proof that the disease is etiologically distinct from human diphtheria.

When one examines microscopically a cover-glass preparation made from the false membrane which forms in a fowl or pigeon, one is struck with the great variety of bacteria present—micrococci, short and long bacilli, as well as numbers of mould fungi and protozoa. The diversity of these organisms, and their more or less frequent occurrence according to the case, explain the great diversity of opinion with regard to the etiology of the disease.

Most authors who have wished to demonstrate the specific nature of a particular bacterium found by them in the lesions have succeeded in experimentally producing false membrane by excoriating the mucous membrane of the pharynx in healthy animals and rubbing some culture of the organism in question into the wounded surface. There is, however, nothing convincing in such a result, since a variety of different bacteria may lead to the development of false membranes when they are thus inoculated. This method of inoculation, moreover, never leads to the development of the citron-yellow fibrinous deposits on the pleura, air-sacs, peritoneum, and oviduct, which are characteristic of avian diphtheria. Avian diphtheria is a general disease, and the majority of the organisms which have been regarded as the cause of it have no tendency to become generalised throughout the body.

A different method of procedure has enabled M. Guerin to detect in every case the presence of the same particular bacterium. The method is as follows:—

From the pharynx, pleura, air-sacs, or peritoneum of a diseased fowl sacrificed for the purpose, one takes a small quantity of the yellowish fibrinous deposit characteristic of the affection. Preferably the material is taken from the air-sacs, as in that position it is generally less contaminated with accidental bacteria than the membrane formed in the pharynx. A piece of false membrane thus selected is triturated with a little distilled water in a sterile vessel. A quarter of a cubic centimetre of the liquid thus obtained is injected into the connective tissue of the lower eyelid of a young pigeon. In from ten to twelve hours afterwards the seat of inoculation has acquired a yellowish-white colour, owing to the afflux of a considerable number of leucocytes to the part. The eye is half-closed, and the lachrymal secretion is increased in amount. In the majority of cases the virulence of the material inoculated is not sufficient to cause the death of the experimental bird.

After forty-eight hours the pigeon is killed, and one collects in a sterile tube the membranous production which has been formed on the inner surface of the lower eyelid. From this, proceeding as before, a second pigeon is inoculated. After the third or fourth passage the pigeon dies within twenty-four hours from a disease which has become septicæmic, owing to the increased virulence of the causal agent. This agent, which is always the same, and found in every case, is a bacterium morphologically resembling the bacillus of fowl cholera. The following are its characteristics. Coccobacilli, non-motile, not stainable by the method of Gram, do not liquefy gelatine or coagulate milk. The reaction of milk in which the organism is cultivated is not changed. No growth on potato, no formation of indol, does not alter the tint of litmus agar. The organism is facultatively ærobic or anærobic, and its cultures exhale a special odour. It is further distinguished by being very exacting with regard to the conditions of life, and exhibiting a marked inconstancy of virulence.

Numerous experiments have shown that of all the common domesticated birds the pigeon is the most susceptible to avian diphtheria, and it is by successive passages through this species that Guérin has succeeded in considerably exalting the virulence of the microbe, and in giving it a fixed character. If a pigeon is inoculated under the skin or into the peritoneum with about 5 cc. of such a virulent culture, death takes place in from twenty-four to thirty-six hours. The lesions observed at the *post-mortem* are those of an extremely rapid septicæmia. All the tissues and juices of the body contain a considerable number of the bacteria, but one never observes the macroscopic lesions which are characteristic of the natural disease.

However, Guérin has succeeded in reproducing all the serious localisations of avian diphtheria by allowing the experimental pigeons to become infected by ingesting food or liquid to which virulent cultures in bouillon-serum had been added. If the birds are young or not very resistant, such as pigeons, this method of infection determines in three or four days a very acute septicæmic form of disease. If the animals are older the symptoms vary according to the localisation of the lesions, which in every respect simulate those of the natural chronic disease.

When the birds are fed with infected materials one can observe during the first few days the appearance in the mouth or pharynx of one or several patches of false membrane. Some days later these may be eliminated and disappear, or they may serve as the point of departure of a proliferating lesion, which in some cases terminates in generalisation of the causal agent.

In other subjects one finds extensive lesions of the lungs and pleura. The lungs present foci of caseous pneumonia. A thick yellowish-white false membrane lines the walls of the chest, and sometimes one finds the mucous lining of the air-sacs similarly covered.

However, the majority of the subjects of experiment show after about three weeks areas of necrosis in the liver, the colour of these being at first yellowish-white, and afterwards during the last days of life of a dull green colour.

In exceptional cases the ingestion of the microbe set up peritonitis, with a thick and very abundant exudate containing large numbers of the specific organism. In a small number of pigeons arthritis developed in the scapulo-humeral and humero-radial joints, which lesions are well-known to breeders of carrier pigeons.

The fæces of birds thus infected are virulent, and it is very easy to isolate the organism from them. For this purpose it suffices to take a small quantity of the fæces mixed with a little sterile water, and to inoculate $\frac{1}{4}$ cc. of the mixture under the lower eyelid of the pigeon. As a rule the first pigeon thus inoculated does not die, but after three or four passages the false membrane forms at the point of the inoculation, and from it one succeeds in isolating the causal microbe in a state of purity. The virulence of the fæces explains

why the ocular lesion is so frequent in fowls infected with this disease. Apart from the numerous chances of direct infection, fowls and pigeons inoculate themselves owing to their habit of frequently scratching their eyelids with their feet soiled with excrement.

With regard to the transmissibility of the disease to man, the only authentic observation of the kind is one reported by MM. Loir and Ducloux. These authors in studying avian diphtheria found at a farm an infant suffering from pseudo-membranous laryngitis, and they alleged that the causal agent of the disease in the child was identical with the one which they found in the false membrane of fowls affected with avian diphtheria on the same farm. However, the description of the organism given by these authors indicates that it was one of the colon group of bacilli, representatives of which class are often found in the most various kinds of false membranes.

M. Guerin has himself paid particular attention to this subject, and his experience has convinced him that the disease is not transmissible to the human subject. In certain districts in the north of France the breeding of game-fowls for cock-fighting is very common among the peasants and workmen; and, as is well known, that breed is particularly susceptible to avian diphtheria, large breeders sometimes losing as many as 40 per cent. of the birds from it.

M. Guerin has seen some of these fowls severely affected with avian diphtheria kept in the room used by the family for taking their meals, and observed the children and others feeding out of the hand fowls that were rendered blind by the diphtheritic conjunctivitis; but in spite of this and other opportunities of infection he has never seen any evidence that the disease could become transmitted to children.

Although it thus appears to be justifiable to assert that avian diphtheria is not transmissible to the human subject, it has been stated that the Klebs-Löffler bacillus may co-exist with the bacillus of avian diphtheria in the false membrane found in fowls. Some of the observations made with regard to this point apparently cannot be placed in doubt, but Guerin holds that such cases are exceptional, and that the Klebs-Löffler bacillus isolated in these circumstances has generally been devoid of pathogenic properties, and only with difficulty identified with the bacillus of human diphtheria. Out of sixty-eight examinations which Guerin was able to make, he was able in two cases to isolate from the false membranes of fowls affected with avian diphtheria a microbe morphologically similar to the bacillus of human diphtheria, but it absolutely failed to produce any toxin. That also was the case with the bacillus isolated in similar conditions by Malvoz. Ferre of Bordeaux, who has most frequently found the two bacilli in association, alleges that he has seen in the diseased fowls paralysis of the legs similar to what is produced by inoculation with the toxin of human diphtheria. Guerin has never seen this, and he records that he has treated more than two hundred fowls affected with avian diphtheria by injections of human anti-diphtheritic serum, but with no greater effect than was produced by treating a parallel series of diseased birds with normal serum of the horse. He has tried with encouraging results a method of sero-vaccination intended to protect fowls against avian diphtheria. The principle of this vaccination consists in sensibilising the bodies of the bacteria by mixing them with a sensibilising serum obtained after practising intravenous and intra-peritoneal injection of large quantities of culture into a horse. Such serum, even in very small doses, renders the bacteria capable of being ingested and destroyed by the leucocytes of normal fowls. Cultures thus sensibilised are very well tolerated in the peritoneum. This anti-bacterial serum also possesses the curious property of preventing the growth of the organism of avian diphtheria when a drop of it is added to bouillon serum, which is otherwise the most suitable medium for its growth. This fact cannot be attributed to agglutination, for the serum does not agglutinate cultures already made.—(*Recueil de Méd. Vét.*, Jan. 1903.)

THE RESULTS OF REPEATED INJECTIONS OF TUBERCULIN.

When the reaction to tuberculin is of a doubtful character it is usually recommended to allow two to four weeks to pass before repeating the test, because no reaction is likely to occur on account of the still unexcreted tuberculin in the animal's body.

During a period of six weeks passed in Hvidding as director of the quarantine department, Dr Bartels made in this connection a number of observations which tend to show that in many cases a second injection, six to eight days after the first, will prove successful.

So far as shown by current literature, Bang was the first to point out that in a number of species repeated injections of tuberculin produced a certain tolerance to the material. Of the total number of cattle on a certain experimental estate, first tested by Bang in the autumn of 1892, 20 per cent. failed to react when again tested in the spring of 1893. Four of these animals, slaughtered for control purposes, nevertheless all proved tuberculous, three to a slight extent and one very extensively.

Nocard also carried out extensive experiments with the idea of solving this question. He found that when the injection was repeated after an interval of one to two days only one-third of the animals reacted, when after eight days not more than half reacted, but when fourteen days were allowed to elapse about two-thirds of the animals reacted. An interval of twenty-five to thirty days after the first reaction was required in order to insure a typical reaction occurring on the second injection.

A. Eber, in contradistinction to Nocard, gives very much more favourable figures. Of a series of nineteen cases which had reacted after the first injection, fourteen when again injected forty-eight hours after the first injection reacted to as marked an extent as on the previous occasion.

On the basis of his own observations made in Hvidding, Dr Bartels also concludes that the failure to react on a second injection made soon after the first is by no means so probable as is generally believed. Before describing his conclusions he gives the following particulars regarding the management of the tuberculin test in the quarantine stations. The period of detention at the Hvidding establishment and in those at Apenrade, Flensburg, and Altona is ten days. During the spring and autumn of each year a limited number of store cattle, which must not be more than four years old, are imported. The tuberculin test is directed to be made as near the end of the quarantine period as possible, and certainly not before the sixth day after arrival. A rise in temperature of one degree centigrade is regarded as a reaction, though in animals which before injection had shown a temperature of 39.5°C . a rise of a half degree centigrade is considered sufficient, provided always that the abnormal temperature cannot be traced to tuberculous disease. The animals which react are killed and examined at the end of the ten days period, and the flesh is disposed of according to regulation. It is now a well-known fact that most of the animals sent into quarantine are inoculated beforehand. In many animals the fact can be detected, the point of injection behind the shoulder showing a painful swelling. Clinically tuberculous animals are not sent into quarantine. If one refused to admit to quarantine all animals which had reacted to this preliminary tuberculin injection, from 13 to 18 per cent. of the total number imported, and in single drafts as many as 40 per cent., would not come under observation. The injections are said to be made partly by the dealers, but usually by the veterinary surgeons. As soon as the business of injection is over the animals are sent into quarantine, and, allowing one or two days for their transport, are tested for the second time six days after arrival, that is, seven to eight days after the first injection. In

several cases Bartels succeeded in obtaining from the less intelligent drivers the temperature records of the first inoculation. During the import period just terminated the results of inoculation in quarantine were as follows :—

Of the 21 animals comprising the 1st consignment 2 reacted.			
"	25	"	2nd " 1 "
"	59	"	3rd " 4 "
"	11	"	4th " 1 "
"	24	"	5th " 2 "
"	24	"	6th " 2 "
"	68	"	7th " 13 "
"	14	"	8th " 1 "
"	20	"	9th " 4 "
"	13	"	10th " 3 "
"	28	"	11th " 5 "
"	29	"	12th " 4 "
"	15	"	13th " 2 "
"	16	"	14th " 3 "
"	54	"	15th " 4 "
"	28	"	16th " 7 "
"	26	"	17th " 4 "
"	29	"	18th " 11 "
"	27	"	19th " 4 "
"	27	"	20th " 6 "
"	26	"	21st " 2 "
"	22	"	22nd " 3 "
"	20	"	23rd " 6 "
"	7	"	24th " 1 "
"	11	"	25th " 1 "
"	14	"	26th " 1 "
"	9	"	27th " 2 "
"	27	"	28th " 3 "
"	9	"	29th " 1 "
"	21	"	30th " 1 "
"	12	"	31st " 0 "
"	19	"	32nd " 1 "
"	13	"	33rd " 0 "
<hr/>			
Total 768			105

Of 768 oxen, therefore, 105, or about 13 per cent., reacted. On slaughter the results of the reaction were in almost every case confirmed; 97 oxen showed more or less extensive tuberculous changes, and only in 8 cases was nothing macroscopically detected.

As shown by the preceding figures, it would appear that the tolerance for tuberculin cannot be so very great, as in six to seven days after the first injection a considerable number again reacted. Nevertheless, amongst the oxen which were admitted into Germany as not reacting to the quarantine injection, there may have been a number which, though affected with tuberculosis, showed themselves insusceptible to the quarantine injection on account of their having been so well prepared in Denmark. It is held to be important to examine how far the attempts to stamp out tuberculosis by means of the quarantine system have been rendered nugatory in consequence of inoculation practised previous to exportation. At anyrate, from the above facts and figures it is clear that a large number of animals will again distinctly react to a second injection made after the lapse of six to eight days. One must, however, not forget that all these inoculated animals were less than four years old, and that the majority were two to three years only and were oxen—

two circumstances which *a priori* would tend to prevent any extensive spread of tuberculosis amongst the imported animals.

What practical conclusions can be drawn from the above observations? What interval should one observe before again testing a doubtful case? Bartels holds that in all instances in which time is not of prime importance one should adhere to the classic period, and only retest after a lapse of three to four weeks. The circumstances are different when dealing with newly-bought draught oxen, when no special contract has been made on purchase. According to the German Act of Parliament the seller is only responsible for tuberculosis up to the expiration of a period of fourteen days. In such cases one should not test until the tenth or twelfth day, inasmuch as an injection made during the first few days after purchase would give no reliable result, or would only indicate that the seller had already tested the animal. He considers that in many cases failure of the tuberculin injection is of importance in forming a diagnosis.

The author in conclusion draws attention to one point which has some practical bearing on forming a judgment regarding the animal's condition. As a result of studying the injection and reaction records in Hvidding he noted the following: The animals which were inoculated at 10 p.m. were usually watered next morning between the first and second times of taking the temperature, fresh well-water being pumped into troughs from which the animals were allowed to drink as much as they wished. By comparing the temperature records of reacting and non-reacting animals, it became clear that the drinking of large quantities of cold water might produce a fall in temperature of one degree Celsius in healthy animals. Animals whose temperature had already risen (tuberculous subjects), however, or in which it rose later, *i.e.*, during the course of the day, did not show this peculiarity, and even large quantities of water so taken had little or no effect on their temperature. —(*Deutsche Thies. Wochenschrift*, No. 28, 1902.)

THE MODE OF ORIGIN OF VERMINOUS ANEURISM IN THE HORSE.

In a previous article on the anatomy and life history of the palisade worm of the horse, Dr Sticker laid down the principle that the chief factor in the origin of the aneurism is not the change in the tunica intima of the vessel but in the media. He was aware that in enunciating this opinion he was in opposition to hitherto received views, and promised to pursue the subject in a further essay. His second paper redeems that promise.

The present views of authors who have dealt with the question of the origin of verminous aneurism in the horse from a pathologically and anatomical standpoint are more or less as follows:—

The worm which attacks the tunica intima of the artery produces irritation. The intima becomes inflamed, ulcerated, and covered with fibrin. The continued sucking efforts of the worm extend this inflammation to the media, or the parasites bore into the intima and produce canals, partly in the intima itself, partly between the intima and media. The diseased process so set up is of the nature of a deforming arteritis, and is the first step towards the formation of an aneurism of the vessel. The diseased processes, therefore, occur in the following order: Primary acute end-arteritis, thrombus formation, acute meso-arteritis, chronic endo-arteritis, atrophy of the muscular tissue, dilatation and hyperplasia of the intima and adventitia.

A sine qua non for the inflammatory changes in the arterial wall, which extend from within outwards, is, however, the supposition that, just as the fully developed worms adhere to the mucous membrane of the bowel by

suction, so do the larvæ to the blood vessel. The latter condition, however, has certainly never been seen. On the contrary, all scientific observers report that they found the worm embedded within the layers of the thrombus, and in most instances with one end, either the head or tail, or both, lying free in the lumen of the artery. Colin also states:—

“The sclerostoma of these aneurysms, like that of the intestine, lives on blood, but sucks it directly from around itself without attacking the arterial walls.”

Some worms are also found lying free in the lumen of the aneurism. Regarding this Bollinger says:—

“The free existence of worms in the lumen of the aneurism must be regarded as a *post-mortem* condition, as it is quite unthinkable that the worms, if unattached, could maintain their position in the strong blood current of the artery. One, therefore, finds empty worm canals in the inner coats of the thrombus near the free-lying worms.”

In addition, however, Sticker was able, in the article above referred to on the anatomy of the palisade worm, to show that the larvæ were incapable of producing injuries of the intima with their mouth-rosette. It is true that during their period of existence within the aneurism the larvæ become converted into young worms, and exchange the mouth-rosette for lip fringes and a suctorial mouth; but all of this occurs within the larval envelope, and as soon as this envelope ruptures the young worms migrate to the terminal branches of the mesenteric artery, where they produce slightly raised thickenings of the wall of the bowel, in which they undergo further development and become sexually mature.

In what way then does the aneurism originate?

If one examines the walls of a partly-developed verminous aneurism in the horse, one remarks a distinct thickening of the wall, but not the slightest change in the intima. The chief point is that the thickening is produced by a firm material, difficult to cut, which lies near the periphery, and is due to fibro-plastic inflammation of the adventitia. At the same time the vasa vasorum of the diseased portion of wall are obliterated and thrombosed. The media is also changed, although normally only one half to one mm. in thickness, it may now be from 3 to 7 mm. The increase is chiefly due to hyperplasia of the muscular tissue, and to a much less extent to growth of the connective tissue. The muscular bundles, which normally form a circular sheath of regular thickness, now appear as ring-like swellings, separated by channels containing a small-celled infiltrate and numbers of young larvæ from 4 to 5 mm. in length. These worm larvæ are the cause of the inflammatory thickening of the vessel wall. In other aneurisms, particularly in those of man, such thickening is absent, the aneurism wall, on the contrary, being usually very thin.

How do the larvæ attain the arterial wall? In a previous paper, Sticker described a case in which migration from the peritoneal cavity appeared probable. Microscopic examinations undertaken later have shown him, however, that this case should have been otherwise interpreted. The blind canal in which the worm was found proved to be a thrombosed nutritive vessel of the arterial wall. Having become cautious through this discovery, he undertook careful microscopic examinations of several aneurisms. The reconstruction of the parts which he was able to make by using serial sections, justified him in concluding that the larvæ found there got into the wall of the artery by means of the vasa vasorum, and that the first effect of their presence was to produce hæmorrhagic infarction of a certain area of the muscle. The next consequence was cellular infiltration of the neighbourhood. Migration of the larvæ, therefore, is followed by processes in the arterial wall similar to those already described in connection with the wall of the bowel.

Once the larvæ attain a size of 8 to 10 mm., they press forward from their seat

between the muscular fibres towards the lumen of the vessel. The hitherto uninjured intima now appears raised in bladders, just beneath which the larvæ are found. In one case he saw ten of these together. The muscular tissue was greatly hypertrophied, to such a degree in fact that it formed transverse bands as thick as a quill, between which were deep grooves filled with masses of small cells containing the empty worm canals.

As soon as the loosened intima has become thinned, and to a still greater degree when the bladders have burst, it becomes covered with a material deposited from the blood and consisting of leucocytes, blood plates, and fibrin. One portion of the main vessel had become dilated to the size of a walnut, and within this cavity the intima was loosened over an area the size of a shilling. The loosened membrane was covered with a soft thrombus. Three larvæ were found under the intima. In another case the intima showed three small necrotic spots leading to excavations the size of a grain of linseed. These excavations were filled with a soft mass containing spindle-shaped cells and elastic fibres. The adventitia was thickened by the formation of firm, white, connective tissue. In the slight deposit of fibrin covering the intima a single female larva of *sclerostoma bidentatum* was found.

According to these observations, the disease process consists at first in loosening of the intima from the subjacent media; the endothelium, fibrous coat, muscular coat, and elastic coat being elevated all together so as to form a kind of bladder. In the meantime the adventitia and muscular coat show marked changes. The opposite condition of affairs, *i.e.*, endo-arteritic changes, unaccompanied by meso-arteritis and periarteritis, was not observed.

The cause of the loosening of the intima is the efforts of the larvæ to penetrate from the inside of the wall towards the lumen of the vessel. Once the intima is irritated and inflamed a thrombus forms in the lumen of the vessel, and attains a size dependent on the degree and extent of the endo-arteritis, being sometimes a thin membraniform deposit of fibrin, sometimes an organised, obturating, or canalised thrombus.

For the series of changes which occur in the arterial wall and in some cases lead to the formation of an aneurism, the term arteritis deformans, used by some authors, was not inapt, had it not been for the fact that it was already identified in human medicine with an entirely specific process, *not* caused by the presence of a worm, and *not* attended by the changes above described. The terms arteritis deformans or nodosa (Virchow), arterio-sclerosis (Lobstein), or atheromatous arteritis, are applied to an exceedingly common disease in man, which most commonly effects the aorta, and in which the most striking changes occur in the intima. These changes consist partly in hardened thickenings, but partly in retrogressive changes in the thickened portions of the intima. The other coats, particularly the media, become secondarily diseased. The various phases are:—

(a) Thickening ("plaque formation") in the intima, sometimes soft and gelatinous in character, sometimes fibrous, often hyaline and firm like the cornea, or like bluish-white or yellowish-white cartilage.

(b) The "plaques" may soften and undergo degeneration (atheromatosis in the stricter sense). Fatty degeneration and occasionally mucoid changes occur. Where the surface undergoes degeneration ulcers form; where, however, degeneration occurs in the depths an atheromatous centre, or so-called atheromatous abscess, forms.

(c) Portions of the intima which have undergone hyaline degeneration may become softened or calcified.

From the above it will be noted that the first and last changes occur in the intima, whilst in verminous aneurism in the horse the intima only becomes diseased secondarily, and in young animals may entirely regain its normal condition. Only in aged animals and in cases where the verminous invasion

has been repeated from year to year, so that the muscular tissue and adventitia have suffered great changes, does the intima show permanent disease like calcification.

The causes of arterio-sclerosis or arteritis deformans in man are stated to be as follows:—

(1) Old age. The typical arterio-sclerosis usually develops after 45 or 50 (senile arterio-sclerosis).

(2) Blood changes produced by intoxications—alcohol, tobacco, lead, and uric acid salts; or infections like syphilis, typhoid, scarlatina, diphtheria, or influenza (juvenile or presenile arterio-sclerosis).

(3) Hypertrophy of the cardiac muscle consequent on severe work—seen in sawyers, sailors, athletes, and rowers.

From the etiological standpoint, therefore, the arteritic changes produced by the larvæ of *sclerostoma bidentatum* can scarcely be included under the same heading as those which occur in arterio-sclerosis of man. In man the commonest form is senile arterio-sclerosis. In the horse 92 per cent. show the arterial changes, and the degree of hardening depends more on the number of the immigrated worms and on the repetitions of the invasions than on the age of the subject.

Regarding the second class of causes, the larvæ appear to irritate mechanically rather than chemically. As to the possibilities of infection, any variety of organism might enter from the bowel or from the outer world. To clear up this question, Sticker made numerous attempts to cultivate from the tissues of the aneurisms and from the small-celled infiltrate of the verminous swellings, selecting portions both from the arteries and from the wall of the bowel, but he has never produced any growth.

Summarising the results of the above investigations, the author finds that the first changes in the arterial wall after the entrance of the larvæ of *sclerostoma bidentatum* are the production of embolic infarcts in the tunica media. Acute mesoarteritis, fibroplastic periarteritis, acute endoarteritis, and slight thrombus formation follow. Further development and emigration of the worms cause extensive losses of substance in the media and intima, marked thrombus formation, hypertrophy of the muscular tissue, and dilatation of the vessel wall. The repeated immigration of new larvæ produces chronic hardening of the adventitia, media, and intima.—(*Ibid.*).

THE OCCURRENCE OF *TRICHINA SPIRALIS* IN THE BADGER.

LÜBKE has published an interesting observation with regard to the occurrence of the *trichina spiralis* in the flesh of the badger. That animal is rather common in the forest regions of East Prussia, and its flesh is often consumed by the foresters and others. Hunters and foresters believe that this ought not to be done without circumspection, since the flesh sometimes harbours *trichinæ*; and, as a matter of fact, that opinion is well-founded, for a number of indubitable cases of trichinosis of the badger have already been recorded in veterinary literature. There is also the well-known case of the professor at an academy for forestry who contracted the disease through consuming badger's flesh by way of trial.

The case observed by Lübke himself was as follows:—

A forester killed two badgers in the woods, and at the request of several workmen there he handed over to them the animals to be used as food. However, before the flesh of the animals had been consumed the possibility that *trichinæ* might be present in the flesh occurred to the forester, and he therefore had it examined by Lübke, who found that *trichinæ* were numerously present in the flesh of one of the badgers. In a piece of muscle about

the size of an oat-seed he found as many as thirteen worms. In several instances two adult worms were found rolled up in one capsule. The capsules were well formed, but had not yet attained their maximum degree of development.

In view of the mode of life of the badger the transmissibility of the disease to that animal is easily understood. According to Brehm, the badger is not above consuming animal offal, and a number of foresters informed Lübke that badgers not infrequently eat mice and rats.—(*Zeitschrift für Fleisch und Milchhygiene*, January 1903).

THE RELATIONSHIP OF VACCINIA TO SMALL-POX.

THE recently published experiments by Dr Copeman afford the strongest possible support to those who have long maintained that vaccinia is simply a modified form of human small-pox. As Dr Copeman observes, the question is one of more than academic interest, for the alleged non-identity of vaccinia and human small-pox is frequently brought in as an argument to discredit Jennerian vaccination.

Jenner himself believed that cow-pox, whether carried through the horse as an intermediary host or not, was originally derived from small-pox in the human being; but, as Dr Copeman points out, a great deal of the small-pox which was prevalent in Jenner's time was of the comparatively mild variety which under the name of "inoculated small-pox" was intentionally produced on human subjects with the object of thereby conferring protection against a subsequent attack by the disease in virulent form. At times the results of inoculations were so mild that no obvious effect was observed, with the exception of the vesicle at the point of insertion of the small-pox virus, and the patient suffered but little inconvenience. It may therefore be assumed that many of them would be capable of following their ordinary vocations, which would hardly be possible in the case of persons contracting small-pox in the ordinary way. Not only were the effects following on cases comparatively mild, but the disease in this form was intentionally brought into many country districts which otherwise could not have become affected. These considerations led Dr Copeman to believe that it was probably from the inoculation form of small-pox, rather than from the ordinary variety, that much of the cow-pox in pre-vaccination times was derived. It is easy to understand how cracks so often found on the udders of cows might become infected by a milker with fingers contaminated by contact with the inoculated sore on his arm.

Dr Copeman therefore determined to put the matter to the test of experiment, and, learning that in Nubia and in certain parts of India the inoculation of small-pox is still carried out, he made numerous attempts to obtain the necessary material, but without success. In default of inoculated small-pox in the human subject, he resolved to try the monkey, which his previous work had shown him to be readily susceptible to the disease, the various phases of which in that animal closely resemble those observed in man. The experiments were commenced in 1898, and the small-pox material was obtained from cases coming under observation during outbreaks of the disease at Middlesborough, Glasgow, and London. In each of three separate series of experiments the human small-pox lymph or pulp was first inoculated direct on calves, but in every instance, so far as could be observed, with altogether negative results. With monkeys, however, success was invariably obtained; and when, after one or more passages through this animal, the contents of the local inoculation vesicles were employed for insertion on the calf an effect was produced which after one or more removes in that animal was indistinguishable from typical vaccinia. Moreover, from the contents of

vesicles raised by this method on the calf children were in turn vaccinated, and a number of these were afterwards kept under observation for about a couple of months. Every such vaccination "took" normally, and in no case was any bad result subsequently observed, either by Dr Copeman or by the parents of the children. In no instance did the eruption become generalised.

The point of interest in these experiments lies in the fact that, whereas human small-pox material employed could not be got to "take" directly on the calf, nevertheless the results typical of ordinary vaccination were obtained when the strain of lymph, after passage through a series of monkeys, was again transferred from the inoculation vesicles of that animal to the epidermis of the calf.

In concluding the account of his own experiments Dr Copeman adds that they have recently been corroborated by results obtained in Burmah, where lymph from the vesicles of cases of human inoculated small-pox has been employed to inoculate calves, and has thus originated on several different occasions strains of excellent vaccine lymph.—(*Medico-Chirurgical Transactions*. Vol. LXXXV).

SOME STATISTICS REGARDING SARCOSPORIDIA.

In connection with the examination of pork for trichinæ, Bergmann gives some notes on the occurrence of sarcosporidia in the swine slaughtered in Stockholm and examined in the meat inspection department there. He found that the commonest seat for sarcosporidia was also that for trichinæ. The greatest number of sarcosporidia in pork were found in August, September, and October, viz., 47·39, 83·88, and 36·15 per cent. in the southern regions, and 30·25, 29·82, and 34·75 per cent. in the northern regions controlled by the bureau. The average percentage during the year was 30·62 and 27·59 for the two districts respectively.

The mode of entrance of the parasite is not known, but from the Stockholm statistics it would appear that the early summer is the most favourable time for invasion with sarcosporidia. Of 25 pigs reared in a well-managed "piggery" near Stockholm all were found affected with sarcosporidia, 18 with tuberculosis, 2 with trichinæ, and 1 with tuberculosis and trichinæ. The writer also examined 29 rats found in the slaughter-house of this establishment and its neighbourhood, and discovered sarcosporidia in 2 and trichinæ in 1.

Of 9 old horses killed for dissection purposes 4 were affected with sarcosporidia, which were found in the inner and outer muscular layers of the œsophagus. A few were found in the diaphragm. They measured $1\frac{1}{2}$ cc. in length and ·75 mm. in breadth.

The œsophagus was examined in 166 slaughtered oxen; sarcosporidia were found in 28 cases, that is to say, 16·86 per cent. They were somewhat rare, not more than 7 being found in any one œsophagus. They had invaded both the outer and inner muscular layers. They measured from ·7 to 7·7 mm. in length.

Bergmann failed to discover sarcosporidia in the œsophagus of sheep, although he carefully examined 111 animals.

In sheep the parasites are known to be so large that they could scarcely be overlooked. Wasielewsky's statement that they regularly occur in sheep therefore requires to be regarded with some reserve. Perhaps their occurrence depends on the particular time of the year.

Bergmann also found sarcosporidia in the œsophagus of a deer. The parasite resembled that of the ox but was more slender. It was, however, present in such numbers that the œsophagus appeared striped with white.—(*Zeitschrift für Thiermed.*, vol. VI., and *Deutsche Thiermed. Wochenschrift*, No. 4 1903.)

ON THE CONSTANT OCCURRENCE OF PATHOGENIC MICRO-ORGANISMS, AND SPECIALLY OF THE ROTHLAUF (SWINE ERYSIPELAS) BACILLUS IN THE TONSILS OF SWINE.

UNDER Olt's supervision Bauermeister examined the tonsils of more than 140 pigs with the object of detecting in them the presence of micro-organisms. He precedes the very full description of his investigations by remarks on the anatomical and histological structure of the soft palate and the tonsils. On carefully examining the gland secretions, Bauermeister found numerous saprophytic bacteria, but in addition several pathogenic forms, amongst them the bacilli of Rothlauf (swine erysipelas). In order to isolate the latter and to test their virulence, Bauermeister used the secretions from the tonsils to inoculate mice, rabbits, and pigeons. Attempts to cultivate the Rothlauf bacillus direct from the tonsils failed, as the other bacteria always "swamped" the growth.

In addition to the bacillus of Rothlauf, Bauermeister frequently found ovoid bacteria corresponding morphologically and culturally with the bacilli of swine plague as described by Loeffler, Schütz, and Preisz. Their occurrence in the nasal mucus and in the mouth and the pharynx of healthy swine had already been recognised by Bang, Jensen, Kitt, and others. It is therefore scarcely remarkable that they should also be found in the pockets of the tonsils of the swine.

In addition to the above, the wide gland-ducts were very frequently found to contain pus bacteria, streptococci, and staphylococci, as well as the bacterium coli communis, the bacillus of oedema, sarcinæ, and other saprophytes.

After indicating the part played by the above-mentioned pathogenic micro-organisms in primary diseases of the tonsils, and the complications rendered possible by the presence of the other saprophytic bacteria, Bauermeister closes his work with the following *résumé*, representing the most important results of his investigations regarding the Rothlauf bacillus. Bacteriological examination of the tonsillar secretion in 140 swine shows that this material always contains bacteria, particularly the Rothlauf bacillus. Certain tonsils which contained great numbers of the Rothlauf bacilli also exhibited a local disease apparently caused by this particular bacillus. It therefore seems possible that from such local centres the Rothlauf bacilli may pass into the lymph and blood circulation, and produce general disease and death. An animal so infected can, in turn, infect the entire herd. This supplies an explanation for the sudden outbreak of Rothlauf in otherwise healthy stocks where no other method of infection appears possible.—(*Archiv für wissen. und prakt. Thierheil.*, and *Deutsche Thier. Wochen.*, No. 24, 1902.)

THE TREATMENT OF TETANUS BY INJECTIONS OF BRAIN EMULSION.

WASSERMANN has shown that brain emulsion acts on tetanus toxin by fixing it, and that this action is limited to the cells. It occurs, however, only after removal of the cells from the body. Fiebiger first worked with rabbit brains, but afterwards chiefly used the brains of lambs. The brain of a recently slaughtered lamb is rubbed down with physiological salt solution in a glass vessel, using a glass pestle, and observing antiseptic precautions. Fiebiger gives about one half litre of the emulsion. This is injected under the skin or panniculus.

Of 25 cases of tetanus occurring in the Vienna clinique, 20 were treated with the injection, the remaining 5 being regarded as incurable. Of the 20 horses so treated, 11 recovered, and 9 died. One horse received two, and another three, injections. The remainder only received one injection. In addition to this general treatment, the point at which the tetanic infection had taken place also received attention. Great care was taken in feeding the animals, an œsophageal tube being used. Those animals which seemed incapable of standing were slung. Apart from Glauber's salts, drugs were not employed. The stable was darkened, and all noise avoided.

At the point of injection, very troublesome abscesses usually occurred, and in some cases extensive suppuration set in, weakening the animal greatly, and necessitating prolonged after-treatment.

Fiebiger believes that injections with brain emulsion constitute as efficacious a mode of treating tetanus as any other, and states that this method is cheaper than using anti-toxin. Its disadvantages consist in the difficulty of rubbing down the brain substance, the inconvenience of injecting so large an amount of the emulsion, and the accessory disease at the point of injection. Nevertheless he feels encouraged to further test it.—(*Zeitschrift für Thiermed.* Vol. VI., and *Deutsche Thier. Wochenschrift*, No. 40, 1902).

THE TREATMENT OF HÆMOGLOBINURIA IN THE HORSE.

SEITZ describes two cases of the disease in which he successfully employed bromide of soda and sugar. In both cases the animals had been rested for several days previously, and fell ill during work. With some trouble they were got back to the stables, where they at once lay down. Before his arrival they had made a few unsuccessful attempts to rise, but could only lift the front part of the body, the hind extremities being paralysed. They sweated profusely, and breathed heavily. Each was given a deep straw bed. The first case received 70 grains, the second 50 grains, of bromide of soda, dissolved in one litre of water. Until the dose was ready for administration, the animals were sharply rubbed with straw wisps. The catheter was passed, and some coffee-coloured urine was drawn off. This urine deposited no precipitate, even after standing for twelve hours. Half an hour after receiving the first dose the animals were given one pound of raw sugar dissolved in water. This was repeated four times a day. Twelve hours after the first dose a second dose of like amount of bromide of soda was given, and the doses of sugar were continued as before. After a further twenty-four hours the urine appeared somewhat lighter in colour. The bromide of soda was therefore discontinued, but the sugar was still given. Thirty-six hours later the urine, when drawn off, appeared muddy. At this stage two strong men were then placed at each animal's tail, with orders to assist it in rising, and with a little help the horses were got up, and found able to stand. They showed no signs whatever of paralysis of the hind quarters.

Needless to say, the animals were frequently turned during the course of the disease, the skin was rubbed from time to time, and suitable diet was provided. Although a considerable interval had elapsed, there was no sign of a return. The treatment is based on the theory enunciated in 1901, to the effect that in this disease there is a great loss of glycogen, which can be replaced by freely supplying sugar.—(*Wochenschrift für Thierheilkunde*, No. 5, 1903).

MILK AND OTHER FOOD POISONING AND EPIDEMIC DIARRHŒA.¹

At a meeting of the Epidemiological Society on 12th December last, Professor Sheridan Delépine read a paper on the bearing of outbreaks of food poisoning upon the etiology of epidemic diarrhœa, with special reference to diarrhœa produced by contaminated milk. The paper was discussed at a meeting on 16th January.

Professor Delépine said that by epidemic diarrhœa was generally meant an infectious disease, the most constant symptom of which was diarrhœa, affecting a number of persons at the same time, more especially during the warm season. One of its special features was its tendency to occur in certain poor populous districts, where it caused a great mortality among children. After discussing the symptoms as described by Dr Ballard in his report, and observing that the difficulty of diagnosis in isolated cases was very considerable, he stated that the *post-mortem* lesions were not characteristic. He then briefly reviewed the bacteriological investigations which had been published, and observed that, though the subject was by no means clear, the bacteria suspected on good grounds to be the cause of outbreaks of summer diarrhœa belonged chiefly to types which were the inhabitants of the alimentary canal; the virulence of several of these microbes had been shown to be capable of considerable variation. The bacilli of the colon group appeared to be most intimately connected with the epidemic diarrhœa both of adults and children. Though these bacilli resembled each other closely, yet when the colon bacilli obtained from the intestinal contents of a number of cases of diarrhœa were subjected to the series of tests usually employed for diagnostic purposes it was found that one or more of the reactions associated with the bacillus coli communis of Escherich were frequently absent. The resemblance between many outbreaks of food poisoning and epidemic diarrhœa was very great, and would be even closer if certain special forms of food poisoning, such as botulism, were excluded. Under the term "botulism" Van Ermengen had described a state brought about by the ingestion of various articles of food, such as ham, tinned or preserved foods, oysters, mussels, etc. It was characterised by comparatively slow onset (twelve to twenty-four hours after infection) secretory troubles, paralysis of certain muscles, constipation, retention of urine, and absence of fever; he had found these symptoms produced by a bacillus which he had named bacillus botulinus. While there was good evidence that some outbreaks of food poisoning were due to ingestion of the flesh of animals suffering from certain forms of septicæmia, enteritis, or pneumo-enteritis, whose flesh owed its noxious properties to various bacilli resembling more or less closely the bacillus enteritidis of Gærtner, Professor Delépine held that the mode of infection was usually different. The most common forms of food poisoning produced symptoms closely resembling those of epidemic diarrhœa; outbreaks of food poisoning might occur at any time of the year, but were most frequent at the time when epidemic diarrhœa was most prevalent. That they affected adults as well as infants appeared to be due chiefly to the kinds of food which produced them; certain foods, such as milk, which were partaken of both by adults and infants, gave rise in both to attacks of diarrhœa similar to typical attacks of epidemic diarrhœa of unknown origin. On the supposition that epidemic diarrhœa was generally the result of a more widely disseminated and less massive form of bacterial infection of food than in the

¹ Reprinted from the "British Medical Journal."

more definite outbreaks of food poisoning, it ought to be possible to utilise the occurrence of certain outbreaks of food poisoning for the purpose of elucidating the etiology of epidemic diarrhoea. With this object he had for the past eight years investigated the bacteria which had given rise to outbreaks of food poisoning, and also the pathogenic properties of the cow's milk supplied to town, which was responsible for much of the infantile mortality due to epidemic diarrhoea. The food poisoning outbreaks had included instances due to milk, cheese, pork pies, tinned salmon, and other foods, while the pathogenic action of over 2000 samples of milk had been examined. By comparing the properties of bacilli obtained from noxious articles of food with those from animals suffering from the effects of inoculation with various samples of milk, he had been able to satisfy himself that these bacilli, with a few exceptions, belonged to the colon group. Professor Delépine then related the circumstances of an epidemic of diarrhoea at Victoria Park, Manchester, in 1894, due to the consumption of milk. Although this outbreak was attributed at the time to the milk from a cow suffering from disease of the udder, Dr Delépine had come to the conclusion that the infection of the milk was more probably due to pollution with excreta of a specially virulent kind. Among the three varieties of bacilli of the colon group found in the milk one which had most of the characteristics of the bacillus enteritidis of Gærtner was pathogenic, a single loopful of pure culture on agar producing death in a guinea-pig within twenty-four to thirty hours. That faecal contamination of milk frequently occurred could easily be ascertained by inspection of badly-kept dairy farms, and by the microscopical and bacteriological examination of milk. It was indeed difficult to conceive how slight faecal pollution of cow's milk could entirely be prevented under ordinary conditions, though this contamination was apparently without serious effects. But when some animals in the herd were affected with intestinal inflammation, virulent bacilli must frequently escape from their bowels, and infect a portion of the milk more or less directly when that fluid was not collected under conditions of strict cleanliness. Preliminary investigations made in 1896-97 showed that milk coming from a distance and transmitted without special precautions for keeping it cool was infectious in a large proportion of cases; investigation was therefore directed subsequently to the examination only of specimens collected in sterilised bottles and packed in refrigerating boxes. Of the samples taken before the refrigerating system was instituted, 10·7 per cent. were pathogenic to animals, whereas after refrigeration was instituted the percentage fell markedly, the highest percentage in any year being 3·6 and the lowest 1·14. The difference would have been still greater if the milk had invariably been cooled at the farm immediately after the milking of the cows, and the high percentage of 3·6, which occurred in 1900, was found to be in great part due to the fact that the veterinary inspector had got into the habit of keeping a number of samples over night before delivering them in the morning. Investigations were undertaken for the purpose of comparing the effects produced by mixed milk generally collected at railway stations with those produced by milk obtained from single cows affected with disease of the udder. When cases of tuberculous infection were excluded it was found that the fatal infection produced in as many as 19·2 per cent. of the animals inoculated with milk could be attributed to disease of the udder in a very few, if any. The conditions which favour the development of infectious properties are illustrated by the following tables: ¹

¹ These tables are extracted from the "Journal of Hygiene," Vol. III., No. 1, 1903, where Professor Delépine's paper is published in full.

TABLE I.—*Mixed Milk coming from a distance of over 40 Miles, and generally kept for from 24 to 60 Hours, and even longer in a few cases. (Tuberculous Samples excluded).*

Mean Temperature Fahr. in the shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
30° to 35°	7	5	12	58·0
35° to 40°	7	11	18	38·5
40° to 45°	2	3	5	40·0
45° to 50°	1	4	5	20·0
50° to 55°	—	—	—	—
55° to 60°	0	2	2	0·0
	17	25	42	39·0

TABLE II.—*Mixed Milk coming from a Short Distance (generally under 20 Miles), most of them kept for Less than 10 Hours (with the exception of 5 out of 7 Bad Specimens, and 4 out of the 22 Good Specimens which had been kept somewhat longer). (Tuberculous Samples excluded).*

Mean Temperature Fahr. in the shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
50° to 55°	1	0	1	100·00
55° to 60°	8	1	9	88·80
60° to 65°	11	4	15	73·20
65° to 70°	—	—	—	—
70° to 75°	2	2	4	50·00
	22	7	29	75·68

TABLE III.—*Unmixed Milks kept for various Lengths of Time, but collected from the Udder in Sterilised Vessels. (Tuberculous Samples excluded).*

Mean Temperature Fahr. in the shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
35° to 40°	6	0	6	100·0
40° to 45°	3	2	5	60·0
45° to 50°	5	2	7	71·5
50° to 55°	—	—	—	—
55° to 60°	—	—	—	—
60° to 65°	0	3	3	0·0
	14	7	21	67·2

The influence of time was shown by the number of specimens remaining good even in warm weather, when the milk had been kept only half a day, but Professor Delépine pointed out that what was produced in a few hours in summer might occur in winter, when the milk was kept for a long time. The baccillus enteriditis sporogenes described by Dr Klein had no relation to the colon group, but was apparently introduced into milk in the same way,

through faecal pollution. In Professor Delépine's experience Klein's bacillus was not so frequently the cause of lesions as the colon bacillus; he had found Klein's bacillus in two samples per 1000, while pathogenic bacilli of the colon group were present in from 100 to 200 per 1000 samples. Streptococci did not seem to be responsible for a greater portion of cases than bacillus enteritidis sporogenes. The facts with regard to the recent outbreak of food poisoning in Derby showed that the infected pork-pies had increased in virulence on being kept, just as would milk when sent from a distance to a town. Professor Delépine concluded, from an examination of all the circumstances, that the infectious properties which food acquires frequently in summer, and which give rise to the ordinary or common type of epidemic diarrhoea, are generally due to bacilli belonging to the colon group, of which the *B. coli communis* (Escherich) and the *B. enteritidis* (Gärtner) were probably two extreme types. He believed that the varieties of most importance were those resembling the bacillus of Gärtner; they did not produce permanent acidity, coagulation, or distinct smell when grown in milk. The absence of acidity and marked smell in milk therefore was not, as generally believed, an index of safety. Such infection of food or milk did not generally lead to serious consequences unless it were massive from the first, or unless the food was kept for a sufficient length of time, and under conditions of temperature favouring the multiplication of these bacilli. Milk, the commonest cause of epidemic diarrhoea in infants, were frequently infected at the farm or through vessels in transit; the preventive measures which Professor Delépine considered to be most clearly indicated were:—

1. Measures securing cleanness of cows, dairy hands, cow-sheds, milk vessels, etc.¹ Similar measures with regard to persons or things coming in contact with any other article of food, manufactured or not. Absolute cleanliness was most difficult to obtain, if not practically impossible. Infection must therefore occur now and again.
2. To guard against the worst effects of accidental faecal infection, the food should be consumed fresh when possible.
3. When the food cannot be consumed fresh, it should be refrigerated—that is, kept at a temperature below 4° C.
4. Where the food cannot be eaten fresh or refrigerated whilst it is kept, it should be thoroughly sterilised—that is, by thorough cooking.

Dr. Newsholme, in opening the discussion, expressed the opinion that the two most important facts in Professor Delépine's paper were: (1) that the bacilli in food poisoning were of the *B. coli* group; and (2) that such milk was pathogenic to guinea-pigs before reaching the consumer, its virulence increasing with the distance the milk had travelled and the temperature at which it had been kept. He had suggested that the infection was due to the faecal contamination, and possibly to enteritis in the cow, in which case the veterinary aspect of the disease became important in relation to prevention. But one link was missing in the chain of evidence; the serum reaction of the blood of patients suffering from infection by the colon-typhoid group of bacilli gave a possible clue to the particular species of bacillus concerned in the infection, and it would have been interesting to compare the serum reaction of the blood of the children suffering from diarrhoea apparently caused by this pathogenic milk with that of the guinea-pigs and of the known varieties of the *B. coli*, especially those allied to the Gärtner bacillus. Lesage, for example, found that the *B. coli* from a child in the acute stage of epidemic diarrhoea was agglutinated by the serum of the same child in 40 out of 50 cases, and the serum of each of these 40 agglutinated the bacilli of the other 39, the reaction disappearing in a few days. According to Lesage the *B.*

¹ See in this connection a paper by W. H. Park (1901), *The Great Contamination of the Milk of Cities. Can it be Lessened by the Action of Health Authorities?* "Journal of Hygiene," Vol. I., p. 391.

coli of normal children were not agglutinated by the serum of those suffering from epidemic diarrhoea, nor did normal serum react on pathogenic forms of *B. coli*. He was glad that Professor Delépine had not found evidence to support the view advanced by Klein that the *B. enteritidis sporogenes* was the cause of epidemic diarrhoea, for it was possessed of so persistent a vitality that it could hardly be destroyed by heat without spoiling the milk as food. He could not, however, accept the view that milk was infected usually on the farm or in transit, though he admitted that the chances of faecal contamination on the farm were very great. He had inquired into the history of 226 fatal cases of epidemic diarrhoea in Brighton in three years 1900-02. In 35 the milk supply could not be traced, but of the 191 in which it was known, 18, or 9.4 per cent., were breast fed, and 84, or 44 per cent., were brought up on condensed milk. The greatest care had been taken to exclude cases in which ordinary cow's milk was given occasionally. In over 53 per cent. of those cases in which the source of the milk was known, the infection must have occurred in the house, unless it were assumed that condensed milk before the tin was opened was pathogenic. In Liverpool, in the third quarter of 1899, over 20 per cent. of the deaths from diarrhoea were of breast-fed children. These figures did not give the relative proportions of children fed on each kind of milk, and it was therefore impossible to say whether cow's milk were more productive of diarrhoea than condensed or mother's milk, but these figures showed either that the infection of the milk did not always take place on the farm or that diarrhoea was caused otherwise than by milk. Professor Delépine's views explained the outbreaks of diarrhoea in which a large proportion of those who partook of these foods were attacked. These milk epidemics resembled those of typhoid due to milk in which every susceptible person who drank the milk was attacked. In such outbreaks the infection doubtless occurred at the farm or dairy. But it was otherwise with the sporadic cases of diarrhoea; in these the cases were distributed over a large number of milk supplies, and Dr Newsholme felt convinced that the milk was infected at the home. He had made an analysis of the 191 fatal cases mentioned above, in which the nature of the milk used could be ascertained, and found that while 88 had had their milk from thirty-eight dairies, 103 had obtained it from other sources; 18 infants were at the breast, 1 was fed on sterilised milk, and 84 on condensed milk. No deaths occurred among the customers of 31 dairies. In the 103 cases the infection must necessarily have taken place at home; the 88 were fairly equally distributed among the larger number of dairies. Again he had divided the dairies into classes according as to whether there were and were not any fatal cases among their customers, and according as the milk was railborne, roadborne, or both, but obtained almost identical results. In short, he was convinced that the home conditions of the urban poor, the storage of milk and food in living or sleeping rooms, and the opportunities for its infection by flies fresh from organic refuse, or the liquid diarrhoea stools of a patient accounted for the prevalence of autumnal diarrhoea quite apart from the infection of milk at its source, which undoubtedly gave rise to epidemic outbreaks of diarrhoea in some instances. Dr Newsholme pointed out that if his contention was correct the reduction of diarrhoeal mortality must be looked for by way of (1) exact instruction, as suggested by Dr Niven, of older girls in day schools in the art of preparing infant's food: in the principles and practice of sterilisation of food and of bottles; and in the minutest cleanliness in connection with the storing and handling of food. (2) Greater domestic and municipal cleanliness, leading to the reduction of dust both in houses, courts, and streets, and the substitution, both domestically and municipally, of wet and more frequent cleansing for the dry and infrequent cleansing now in vogue. (3) A crusade against the domestic fly, which was most numerous at the season and in the years when epidemic diarrhoea was most prevalent, and probably

FROM THE ANNUAL REPORT FOR 1902 OF THE PRINCIPAL OF THE ROYAL VETERINARY COL- LEGE TO THE ROYAL AGRICULTURAL SOCIETY.

RESEARCH LABORATORY.

DURING the year 1902, 404 morbid specimens were forwarded for examination to the Laboratory established at the Royal Veterinary College in 1890 for research in Comparative Pathology and Bacteriology, and which has since been maintained by the aid of an annual grant of £500 from the Royal Agricultural Society. The number of specimens similarly forwarded in 1901 was 488, and in the preceding year 343. As in former years, the material which reached the Laboratory in this way proved of great value in the teaching of pathology and bacteriology, although, as a rule, the specimens are not sent with that object. In the great majority of instances the material is sent for investigation in cases where the disease presents some unusual feature or the cause of it is obscure, and the utility of the Laboratory in that respect has now come to be recognised throughout the kingdom.

RABIES.

The returns with regard to this disease for the past year are distinctly disappointing, since they show that rabies cannot yet be considered as exterminated. Indeed, during the past twelve months more cases of rabies in the dog were detected than in any year since 1898. The following table shows the incidence of the disease among dogs during the past eight years, and in connection therewith it ought to be noted that the practice of imposing Muzzling Orders in all districts in which cases of rabies had recently been detected was begun in March 1897.

<i>Year</i>	<i>Counties</i>	<i>Cases</i>	<i>Year</i>	<i>Counties</i>	<i>Cases</i>
1895	29	672	1899	4	9
1896	41	438	1900	2	6
1897	30	151	1901	1	1
1898	10	17	1902	4	13

With the exception of one case in Devonshire, the disease during the past year has been confined to Wales. All the cases in the dog were reported during the first five months of the year, except the last case for the year, which was detected in the month of December.

SWINE FEVER.

It cannot be said that the figures for the past year hold out much hope that this disease will be entirely stamped out during the near future, although they are very satisfactory as compared with those for the preceding year. The following table shows the number of outbreaks of the disease reported during each year since 1896 :—

<i>Year</i>	<i>Outbreaks</i>	<i>Year</i>	<i>Outbreaks</i>
1897	2155	1900	1940
1898	2464	1901	3140
1899	2322	1902	1688

The substantial reduction in the number of outbreaks during the past year is doubtless the consequence of more energetic action on the part of the Board of Agriculture, especially in tracing the origin of outbreaks by skilled officers, and in slaughtering diseased and suspected animals. It is probable, however, that severer measures than those hitherto enforced will yet have to be taken if the disease is to be stamped out.

It is much to be regretted that during the past year an attempt has been made to sow doubt in the minds of pig-owners regarding the specific nature of what is termed swine fever, and to suggest that the disease may arise independently of contagion or infection, and even be produced by feeding on particular kinds of food. Such a suggestion is obviously calculated to provoke opposition to the measures which are now being enforced with the object of stamping out the disease—measures which would have no justification if swine fever were a disease that could be generated by mere errors in diet. The supposition that swine fever can arise independently of contagion, or be caused by any other agent than the swine fever bacillus, is violently opposed to an immense body of evidence, and the only fact that can be cited in support of it is that in a considerable number of outbreaks such inquiries as are made by the officers of the Board of Agriculture fail to establish a connection with an antecedent outbreak. It is hardly necessary to point out that by the same method of reasoning one might prove that nearly all the diseases universally regarded as purely contagious may arise independently of contagion. To take but a single example, it would have to be admitted that foot-and-mouth disease may be produced by special climatic conditions or improper feeding. It is only reasonable to expect that it will not be possible to trace every outbreak of swine fever to contagion, but experience has shown that the more searching the investigation the larger is the proportion of outbreaks in which contagion can be traced, and that the disease never originates in circumstances that exclude the possibility of contagion.

FOOT-AND-MOUTH DISEASE.

Only one outbreak of this disease was confirmed in Great Britain during the year 1902. This was detected in the month of March last among a flock of 330 ewes with their lambs in the parish of Chislet, in Kent. The farm being in an isolated position, the Board of Agriculture decided not to slaughter any of the animals, and the disease extended throughout the entire flock, causing the death of many of the lambs. Two cattle in an adjoining field belonging to the same owner were also attacked. The measures of rigid isolation which were imposed by the Board proved successful, and since the farm in question was declared free no case of the disease has been detected in the United Kingdom.

In the month of April the disease appeared in a small herd of cattle in the island of Guernsey, and it afterwards extended to the animals on a neighbouring farm, where some human beings were also reported to have become affected. Fortunately, the disease did not extend beyond the limits of these two farms.

Investigation failed to discover the cause of either of these two outbreaks, and, as in both cases the importation of the disease with live animals was excluded, one must suppose that the contagion was introduced by some intermediate agent (human or inanimate) from the Continent of Europe.

In this connection it may be of interest to refer to the equally mysterious appearance of the disease in the United States of America during the past year. Its existence there was first recognised in the month of November, but at that time about 100 herds, scattered over several New England States, were already affected, and it is admitted by the officials of the U.S. Department of Agriculture that the contagion must have been introduced as early as the month of August last.

The first cases appear to have occurred at Chelsea, Massachusetts, not far from the docks; and it is surmised that the contagion was brought with imported hay or bedding for horses. A determined effort has been made to stamp out the disease and to prevent it from spreading westwards, over 2300 diseased or suspected animals having been slaughtered up to 6th January last. It is to be hoped that the attempt to eradicate the disease will be successful, but it is obvious that a considerable period must be allowed to elapse after the last discovered case before concluding with any degree of confidence that the plague has been stamped out, especially in a country where, as experience has shown, it was able to exist for three months unknown to the authorities.

ANTHRAX.

As will be seen from the following table, the official returns for the past year show a slight increase both in the outbreaks and the number of animals attacked with this disease during the past year:—

<i>Year</i>			<i>Outbreaks</i>			<i>Animals attacked</i>
1899	531	986
1900	571	956
1901	651	971
1902	687	1042

A remarkable feature in these figures is the small number of animals attacked in each outbreak, the average being less than two. The fact would appear to indicate that in this country there is very little land seriously contaminated with the germs of anthrax. In many cases only a single animal is lost from the disease in the course of the year, and not rarely a case occurs on a farm where the most searching inquiry fails to bring to light any history of a previous case during the last half-dozen years. It is well known that the spores of anthrax may retain their vitality for a very long period in the soil or elsewhere, and it can therefore be understood that one case may be attributable to an antecedent one on the same farm, although a long period may have separated the two cases.

It is possible, however, that some outbreaks of anthrax, where what appears to be a reliable history indicates previous freedom from the disease, may have another explanation, viz., that the germs of anthrax have recently been introduced to the place with feeding-stuffs or bone manure. Indeed, at the present time there is a very widespread belief that a good many outbreaks are brought about in this way, and there is one class of cases in which there is hardly any room for doubt that the belief is well founded, viz., those that occur among city horses. During the past year a case of anthrax in a horse belonging to a London stud was diagnosed by microscopic examination of portions of the animal which were sent to the Laboratory, and on inquiry it was found that there was a history which pointed with more or less probability to several other deaths from anthrax in the same stud during the previous few weeks. It was ascertained that the oats on which the horses were being fed were of foreign origin, and it is not improbable that these were contaminated with the germs of anthrax, although it was found to be impossible to prove that by experiment.

It appears to be desirable to repeat here a warning that has been given in previous reports. As in former years, a number of outbreaks of anthrax among pigs have come under notice, the affected animals exhibiting the usual symptom of swelling of the region of the throat, and inquiry eliciting the fact that they had recently been fed with the raw flesh of some farm animal recently found dead. It cannot be too strongly impressed on farmers and others that in every case of sudden unexpected death of a horse, ox, or sheep,

anthrax should be suspected unless it is clear that the animal has died from some other cause. Furthermore, the carcase of such an animal should not be skinned or opened, nor should its flesh be used for any purpose.

GLANDERS.

The number of cases of this disease officially reported during the past year was 2073. Twenty years ago, viz., in 1882, the total number of cases reported was 1389. It thus appears that, although during the whole of that period glanders has been a scheduled contagious disease, subject to regulations designed to prevent it from spreading, it is now more prevalent than it was twenty years ago. As will be seen from the following figures, the past year compares favourably with the immediately preceding one, but if 1901 be excepted one has to go back as far as 1893 to find a year in which the reported cases reached 2000.

<i>Year.</i>	<i>Number of Cases.</i>	<i>Year.</i>	<i>Number of Cases.</i>
1893	2133	1898	1385
1894	1437	1899	1472
1895	1594	1900	1858
1896	1294	1901	2370
1897	1629	1902	2073

It has for a number of years been generally recognised that glanders is mainly spread by the agency of horses affected with the disease in the occult or latent stage, and that any attempt to stamp it out is certain to prove a hopeless task unless account is taken of the apparently healthy animals that are known to have been exposed to contagion. It need hardly be said that in this respect glanders is in no way peculiar, for the necessity of dealing with the apparently healthy animals that have been exposed to contagion arises in connection with all the contagious maladies, and effect has been given to this in the regulations that have been framed and enforced to combat cattle-plague, pleuro-pneumonia, foot-and-mouth disease, etc.

In dealing with contagious diseases that run a rapid course, are always manifested by pronounced and characteristic symptoms, and generally terminate in recovery (foot-and-mouth disease for example), temporary isolation may be successfully and economically employed to counteract the danger attaching to what may be called contaminated animals, that is to say, such animals as still appear to be healthy, but are known to have been recently exposed to the risk of contagion. On the other hand, when the disease to be dealt with does not possess these characters, the method of isolation becomes impracticable or useless. It is useless in the case of cattle-plague and pleuro-pneumonia because of the fatal character of these diseases, and in dealing with them recourse was therefore had to the "stamping-out" system, under which the suspected animals had to be treated in the same manner as those visibly affected, and submitted to summary slaughter. The method of isolation is also inapplicable to glanders, not because the disease is invariably or generally fatal, but because the majority of cases of glanders run a slow insidious course, and for a long period after infection are not attended by any symptoms of disturbed health, or at least not by any that are very characteristic. On this account it at one time appeared to be hopeless to expect to eradicate glanders by any less drastic methods than those which had to be enforced

against such diseases as cattle-plague and pleuro-pneumonia. But to have applied the stamping-out system to glanders would have involved an enormous sacrifice of apparently healthy and useful horses, and hence that method of dealing with the disease was never seriously contemplated.

However, the discovery of mallein in the year 1890 put a new complexion on the matter. As soon as sufficient experience had been accumulated to show that this agent could be relied upon for the detection of glanders, even in its early stages, the hope began to be entertained that it might be possible to eradicate the disease without any greater sacrifice than the slaughter of the animals actually affected. Given an outbreak of glanders, all that would be necessary would be to kill the obviously affected horses, test the remainder with mallein, and slaughter those that reacted. The premises having been adequately disinfected, the outbreak would be at an end. Such in theory was the use that could be made of mallein, and in a good many outbreaks in recent years the method above sketched has been voluntarily and successfully put into practice. It has, however, become clear that as a compulsory general method of dealing with the disease it is at present impracticable on the ground of cost, for when the mallein test is applied to a large stud in which glanders has prevailed for years, the number of horses that react is often so great that their slaughter would mean ruin to their owner.

The next step in the development of opinion with regard to the best method of dealing with glanders was the natural consequence of the discovery that the disease is by no means invariably fatal, and that a considerable proportion (sometimes the majority) of the apparently healthy horses that react to mallein fail to develop any symptom of the disease, even when they are kept alive for years after the test. Moreover, the same test repeated after a long interval may indicate that the animals have completely recovered, and *post-mortem* examination may prove that this indication was correct. A knowledge of these facts suggested that outbreaks of glanders might be successfully and economically treated by testing all the suspected but apparently healthy animals with mallein, and subjecting to a modified isolation all those that reacted.

Both at home and abroad this plan of dealing with affected studs has been tried on a considerable scale, and generally with fairly satisfactory results. Obviously, to render the method practicable, the isolation of the apparently healthy horses that have reacted to mallein must not be of the rigid sort applicable to animals suspected of such a disease as cattle-plague or foot-and-mouth disease, for to confine a horse to the stable for a year or more would inflict on the owner a loss almost as serious as that imposed by the animal's immediate slaughter. To make the plan workable, horses that have reacted to mallein but are apparently healthy must be allowed to work on the streets or elsewhere, but within the stable they must be completely isolated from other horses. It is also obvious that this modified isolation would have to be permanent, or at least maintained until a later test with mallein indicated that recovery had taken place.

The majority of those who gave evidence before the Departmental Committee on Glanders (1899) were in favour of this method of dealing with the disease, at least in the case of outbreaks in large studs in London and other big towns, but the Board of Agriculture appears to have been reluctant to give any official sanction to the plan proposed, mainly because it involved the assumption that apparently healthy horses that have reacted to mallein might with safety be allowed to work on the streets. The small amount of weight that ought to be accorded to this objection will be apprehended when it is reflected that at the present time certainly hundreds, and not improbably thousands, of apparently healthy but in reality glandered horses are daily at work on the streets of London. The plan above referred to would not increase the number of such horses at work, or increase the chances of infection in the

streets. The only difference would be that under this method of dealing with the disease the authorities would have more precise knowledge with regard to the number of cases of occult glanders actually in existence.

In the early part of 1901 the Board of Agriculture appointed a second Departmental Committee on Glanders, mainly in order to obtain more precise evidence than was then in existence with regard to the risks involved in allowing horses that have reacted to mallein to associate with healthy animals. With this object, the Committee carried out a number of experiments, which were begun in January 1901, and brought to a close during the past year. In reality two distinct sets of experiments were instituted, these having respectively the following objects:—

1. To ascertain whether an apparently healthy horse which reacts to mallein is capable of spreading the infection of glanders under ordinary circumstances.
2. To ascertain whether an apparently healthy horse that first reacted to mallein, and subsequently ceased to react, is capable of spreading the infection of glanders.

Experiments with "Reactors."—The general plan of these experiments was briefly as follows. In the first place, the Committee had to find a number of horses that had recently reacted to mallein, but which showed no external manifestation of glanders or any symptoms from which it could be inferred that they were affected with that disease. In the second place, they had to obtain a number of horses free from glanders in the sense that they appeared to be healthy and did not react to mallein when tested for the first time immediately before or after purchase for the purpose of the experiments. The necessary number of animals answering to these descriptions having been obtained, the two sets of horses were placed together. The building in which this enforced co-habitation was carried out was, at the time when it came under the control of the Committee, entirely new, and had never been used for horses. At the outset it was divided into stalls by swinging bales, but this arrangement of the interior was altered on 1st March, on which date the whole of the swinging bales and the other internal fittings were removed, so as to convert the building into what was simply a large rectangular shed. A number of troughs were fixed to the walls at a convenient height to serve as mangers, and a common water-trough was also provided. Into the stable as thus altered, both sets of horses ("reactors" and healthy) were turned loose in order to afford the freest play to infection.

The experiments included the testing of the originally healthy horses with mallein after a period of exposure to infection in the experimental stable, and ultimately a *post-mortem* examination of all the horses, coupled with the application of cultural and inoculation tests¹ to any lesions thus discovered, except those that were obviously not of a glanderous nature.

Although it would naturally have been of interest to ascertain how soon after exposure to infection any of the originally healthy animals became diseased, it was thought better not to test these horses frequently with mallein lest it might be said that this had exerted an immunising or a curative effect.

The number of horse employed in this experiment was fourteen, these comprising six "reactors" and eight horses that were presumably free from glanders at the time when they were introduced into the experimental stable. The period during which the healthy horses were thus kept in association with the "reactors" varied from fifteen to fifty-five weeks. The general result of the experiments was that only one of the healthy horses became affected with glanders, the other seven being free from the disease when they were killed. The experiments thus proved, what, indeed, no one had ever doubted, that in practice a horse that reacts to mallein must be considered

¹ This part of the investigation was conducted by the writer in the Research Laboratory at the Royal Veterinary College.

capable of spreading the infection of glanders to healthy horses kept in the same stable.

Although such degrees of contact as are established between horses in the streets or elsewhere at work might be held to come under the head of "ordinary circumstances," the Committee were unable to devise any experiments bearing directly on the risk of infection between "reactors" and healthy horses at work in the open air. At the same time, in view of the fact that in the above experiments only one of the originally healthy horses became infected, in spite of their long-continued close association with a number of "reactors," the Committee were of opinion that the danger of the disease being spread by apparently healthy reacting horses while at work is so slight that in practice it might be neglected.

Experiments with "Ceased Reactors."—In this set of experiments horses that had on one or more occasions reacted distinctly to mallein, but had failed to react on later tests, were turned loose in the same stable with healthy horses. The stable in question was quite new and devoid of any internal fittings. For the purpose of the experiment it was provided with feeding troughs and a watering trough, as in the preceding set of experiments.

The number of horses used in this set of experiments was eleven, five of these being "ceased reactors," and the remaining six presumably healthy at the time of purchase. The period during which the horses were kept in the experimental stable varied from three to fifty-three weeks.

At the conclusion of the experiments each of the animal was slaughtered and submitted to *post-mortem* examination, and, as in the first set of experiments, all the discovered lesions that had even the most distant resemblance to glanders nodules were submitted to cultural and inoculation tests. The results of this experiment were in every case negative. During the course of the experiments none of the animals developed any clinical symptom of glanders, and no evidence of the presence of active glanderous disease was detected in any of the horses when they were killed. This experiment, therefore, indicated that a horse that has ceased to react the mallein is incapable of spreading the infection of glanders. This conclusion, however, is subject to the qualification that there must have been an interval of not less than two months between the last two mallein tests. In the horses that were used in this experiment, the shortest period between the last two tests to which the animal had been subjected before it was purchased by the Committee was ten weeks, but in most of the cases it was considerably longer than that. The Committee considered that in practice the mallein tests should not be repeated oftener than every three months when the object is to ascertain whether the animal has actually recovered from glanders.

The results of these experiments, and of others of the same kind, may be said to have cleared the ground for some modification of the existing regulations with regard to glanders. The alteration that is most urgently needed is to take practical account of the fact that glanders is mainly spread from stable to stable by the sale and purchase of apparently healthy horses coming from infected studs. In other words, when a case of clinical glanders is discovered the stable companions of the visibly affected animal must be considered glandered until this supicion has been removed by the result of a mallein test. If the disease is ever to be stamped out, the horses that react must either be promptly killed, reasonable compensation being allowed to the owner, or they must be submitted to the modified isolation described in the previous pages.

QUARTER-EVIL OR BLACK-QUARTER.

As a disease of young cattle, quarter-evil, murrain, or black-quarter, has for a long time been only too well-known to farmers and veterinary surgeons in

many parts of the kingdom. No precise information as to the number of animals that annually die from it is at present available, but there can be no doubt that the disease is the cause of a very serious annual loss, and in some districts it is the most formidable disease which breeders have to contend with.

For rather more than twenty years the actual cause of the disease has been well known to the members of the veterinary profession. It is a microscopic germ or bacillus, which in every case of the disease is found in countless numbers in the peculiar inflammatory swelling characteristic of the affection. The belief that this bacillus is the cause of the disease is not a mere inference from the constancy of its presence in the inflammatory swellings; it rests mainly on the results of numerous experiments proving that when a susceptible animal is inoculated with these bacilli it becomes infected in precisely the same way as if it had contracted the disease naturally—that is to say, without man's intervention.

Although the actual cause of the disease is therefore no longer in doubt, it would be wrong to represent that everything connected with the origin of the malady is thoroughly understood. It appears to be in the highest degree probable that the bacillus of black-quarter belongs to the class of organisms that are able to multiply and maintain their existence in the soil. Like some other soil organisms, these bacilli appear to have a very unequal distribution at the earth's surface, being numerous in the soil in some districts and unknown in others. No doubt this is the explanation of the fact that quarter-evil is obstinately attached to certain farms, or even to particular fields, while in other parts of the country no case of the disease is ever seen.

The principal point in connection with the causation of the disease that is still very obscure is the manner in which the quarter-evil bacilli gain access to the animal body, or rather to the animal tissues. If these bacilli are in some parts of the country normal inhabitants of the soil, it is impossible that animals grazing there can avoid introducing them into their digestive organs, adhering to food materials or suspended in water. In searching for an explanation of the infection of animals, one would therefore naturally suppose that the disease must be contracted through the accidental ingestion of quarter-evil bacilli, just as cattle and other animals are usually infected with anthrax through taking in the bacilli or spores of that disease with food or water. The chief obstacle to the ready acceptance of this view is the fact that it is practically impossible to experimentally infect an animal with quarter-evil by feeding it even with enormous numbers of the bacilli or spores, although a very small number of the organisms may suffice to kill the same animal if they are injected under its skin or into its muscles.

In spite of this fact, however, some authorities believe that the naturally-occurring cases of the disease are due to infection by way of the intestine, and endeavour to harmonise this view with the failure of feeding experiments by supposing that special conditions of the intestine may be necessary for infection with quarter-evil bacilli, such special conditions being perhaps brought about by other bacilli accidentally present in the alimentary canal. At any-rate, the only alternative view possible is that animals contract quarter-evil through accidental inoculation with soil containing the germs of the disease, perhaps through small wounds or abrasions about the feet. That view would be more in harmony with the results of experiments, but at the same time there are considerable difficulties in the way of accepting it.

Owing to the virulent character of the quarter-evil bacilli and the rapidity with which the disease runs its course, remedial treatment is practically hopeless, and it has been the lot of very few people to see a case of the disease recover. On this account, and because in certain places the risk of infection is unavoidable, all hope of coping with the disease centres in the possibility

of being able to protect animals against it by vaccination or some other process. For many years young cattle in quarter-evil districts have been setoned with this object, but, although the operation is still practised, it is gradually falling into disuse. The sooner it is entirely abandoned the better, for there is not the least reason to believe that it is of any value as a means of protecting animals against this disease. It was founded on entirely erroneous views regarding the cause of the disease, and it appears to have been continued because of the necessity of "doing something," and want of knowledge of anything better. Whether anything better is really known must next be discussed, but, in any case, there can be no justification for continuing an operation that is admittedly useless.

Since 1883 it has been known that it is possible to protect animals against quarter-evil by a process of inoculation or "vaccination," and during nearly the whole of the time that has since elapsed the operation has been practised on a very large scale on the Continent of Europe, especially in Switzerland.

The "vaccin," or material used to confer protection, is prepared by taking the fresh muscular tissue from the inflammatory swelling of a natural or experimental case of quarter-evil in a sheep or young bovine animal, drying it in a low temperature, and then heating it in order to weaken or attenuate the virulence of the spores which it contains. These spores are remarkable for the resistance they offer to heat, provided the material containing them has first been dried at a low temperature, and experience has shown that they are not entirely destroyed by many hours' exposure to the temperature of boiling water (100° C.). In the preparation of what is called the first vaccin, the material containing the spores, taken as stated above, is exposed for seven hours to a temperature of 100° C. In the preparation of the second vaccin the procedure is the same except that the temperature employed is 90° to 95° C. The first vaccin is, of course, the weaker or less virulent, and it is employed in order to confer such a degree of protection as will enable the animals to bear the stronger second vaccin, which is mainly relied upon to confer the protection that will afterwards enable the animals to withstand any risks of infection to which they may be exposed while at pasture or elsewhere. A very extensive experience, has shown that the safest place at which to inject the vaccins in cattle is near the tip of the tail, the risk of accident in consequence of the vaccin proving too strong being somewhat greater when the operation is performed before or behind the shoulder.

There is absolutely no need for any further evidence as to the merits and demerits of this method of attempting to vaccinate young cattle against quarter-evil, since there are available trustworthy statistics regarding the vaccination of more than half a million animals in this way. In the first place, there appears to be no room for doubt that the operation when properly carried out confers a really valuable degree of protection against quarter-evil, deaths from that disease being highly exceptional among vaccinated animals during the twelve months following the operation, even on pastures known from previous experience to be highly dangerous.

Unfortunately, one considerable drawback attaches to the operation, viz., the risk that the vaccin may prove to be too strong, in spite of the exercise of the greatest care in its preparation. In Swiss statistics relating to the vaccination of 499,000 animals, only one animal out of each 1470 inoculated died in consequence of the operation. This statement is apt to convey the impression that the operation is practically devoid of risk, but in order to enable any one to gauge the danger of the vaccination, it ought always to be explained that the accidents that follow it are very unequally distributed. If, for example, some hundreds of lots of young cattle are vaccinated, the number in each lot averaging twenty, the chances are that only one or two of the owners will experience any loss, but the one or two unfortunate owners may lose as many as 10 per cent. of their vaccinated animals.

It may be thought that even when stated in this way the risk is so small that in practice it might be neglected. That, however, is not the case in the ordinary conditions existing in this country, because the professional discredit experienced by a veterinary surgeon who has had the misfortune to lose several animals in one herd after vaccination is very serious, and cannot be adequately expressed by stating how small was the percentage of his losses. Moreover, reports of such unfortunate accidents naturally obtain great publicity in the neighbourhood, with the natural consequence that no one has afterwards the courage to submit his animals to the operation. During the past year 627 doses of vaccin prepared in the Research Laboratory at the Royal Veterinary College were issued for the vaccination of young cattle in different parts of the kingdom, and, so far as is known, the number of animals that died in consequence of the operation was three. Unfortunately, the whole of these occurred on one farm, where the total number of animals vaccinated was eight. The merits and demerits of the vaccination may be summed up by saying that it is the only known method by which young cattle can be protected against quarter-evil, that the risks attaching to it are such that it ought not to be carried out on farms where the annual loss from the disease is small, but that these risks may well be faced where, on the basis of past experience, one may calculate with tolerable certainty that several animals will die during the year from quarter-evil if vaccination is not practised.

Quarter-evil among Sheep.—As a disease of sheep, quarter-evil has hitherto received very little attention, either in this country or abroad. It has long been known that the sheep is even more readily infected than young cattle by inoculation, and hence it might reasonably be expected that cases of the disease among the ovine species would be of comparatively common occurrence where sheep and cattle are kept on what may be called quarter-evil farms. Such, however, is not the case, if one may judge from the small number of recorded instances of quarter-evil among sheep, but it appears to be possible that a good many cases of the disease in that species pass undetected. At any rate, during recent years investigations made in the Laboratory have on a considerable number of occasions shown that on certain farms serious losses among sheep were ascribable to quarter-evil. A notable instance of this kind was mentioned in a previous report, and during the early part of last year several cases of quarter-evil among sheep came under notice in the Laboratory. As remedial treatment is quite as hopeless in the ovine as in the bovine species, the advisability of having recourse to protective inoculation had to be taken into consideration. Unfortunately, it was found that there was not available for guidance in the matter any recorded experience of vaccination of sheep in this way, and it did not appear to be safe to infer that the operation would be no more dangerous in the case of sheep than in cattle. It was therefore determined to carry out a number of experiments to ascertain whether the previously described "vaccins" could with any degree of safety be used on sheep, and what degree of protection against the disease could thus be conferred.

The first experiment was carried out on five ewes in the month of May last. One or the ewes was set aside to serve as a control animal, that is to say, no attempt was made to vaccinate it. The other four animals were vaccinated as follows:—

No. 1 was inoculated with the first vaccin only.

No. 2 was vaccinated with both first and second vaccin, an interval of twelve days being allowed between the two operations.

Nos. 3 and 4 were inoculated with the second vaccin only.

In each case the vaccin, suspended in sterile water, was injected into the connective tissue between the claws, and the operation did not appear to cause inconvenience to any of the animals. Thus far the experiment was

therefore quite successful, and the next step was to ascertain whether the vaccination had conferred any protection on the animals. With this object the four vaccinated ewes and the one which had been left unvaccinated were tested by injecting a quantity of virulent quarter-evil material into the muscular tissues of the left thigh of each animal. This was done nine days after the last vaccination. On the following day (22nd May) the unvaccinated ewe died from typical quarter-evil. Two days later the ewe No. 1, which had received only the first vaccin, also died from quarter-evil. The other three vaccinated ewes remained well, save that No. 3 was slightly lame for a day or two after the injection of the virulent material into its thigh.

The results of this experiment were thus entirely favourable. It is true that one of the vaccinated animals was unable to withstand the severe test applied to it, but it had only been vaccinated with the first vaccin, which, as previously stated, is only intended to prepare the animals for the second and stronger vaccin. It may also be noted that the survival of this animal till the third day after its inoculation with the virulent material indicated that it had derived some protection from the first vaccin.

On 9th June last two lambs (Nos. 5 and 6) about four months old were vaccinated with the second vaccin only, the liquid being injected into the foot, as in the previous cases. The lambs were not inconvenienced by the operation. On 12th June two other lambs (Nos. 7 and 8) were similarly vaccinated. On 16th July, Nos. 5 and 7 and an unvaccinated lamb of the same age and breed were tested as to their resistance by injecting a small quantity of virulent quarter-evil material into the substance of the thigh of each animal. On the following day the unvaccinated lamb was ill and unable to bear any weight on the inoculated leg, but in the course of a few days it completely recovered. The two vaccinated lambs did not appear to be affected in any way by the test inoculation.

On 21st July, the remaining two vaccinated lambs (Nos. 6 and 8) and a control or unvaccinated lamb were tested in the same way, but with double the dose of virulent material. On the following day the unvaccinated lamb and also No. 8 died from quarter-evil. Two days later (24th July) No. 6 died from an infection which had its starting point in the inoculated thigh, but the *post-mortem* examination showed that this was not caused by quarter-evil bacilli, but by other bacteria which had been accidentally present in the material used for the inoculation on 21st July. It may be observed that the risk of such an accident is unavoidable in using virulent quarter-evil material which has never been heated. Leaving this last lamb out of account, it will be noticed that this experiment was unsatisfactory, inasmuch as one of the vaccinated lambs died from quarter-evil when it was inoculated with virulent material. On the other hand, in fairness to the vaccination, it ought to be admitted that the test to which the lamb was put was a very severe one—probably much more severe than that to which a lamb is exposed when it grazes even on badly infected quarter-evil land.

The results of two other experiments may be stated with less detail. On 27th October last two previously vaccinated lambs, about six months old, and an unvaccinated lamb of the same age, were tested as to their resistance by injecting virulent material into the muscular tissue of the thigh. The unvaccinated lamb died in less than twenty-four hours from quarter-evil, while the two vaccinated lambs were apparently unaffected by the inoculation.

On 8th December last, three previously vaccinated lambs, and one unvaccinated, were tested with virulent materials as in the previous experiments. The unvaccinated lamb and one of the vaccinated ones died on the following day from quarter-evil. The other two lambs were lame after the virulent inoculation, but they soon recovered.

The general result of the experiments may be held to indicate that the vaccination of sheep against quarter-evil might be carried out without greater

risk than attends the operation in young cattle, and that it would be well worth while to give the method a cautious trial on farms where the losses from quarter-evil are serious.

PARASITIC INFLAMMATION OF THE FOURTH STOMACH IN RUMINANTS.

During the past year a considerable number of cases of this disease in sheep (Lincolnshire lamb disease) came under notice, though it does not appear to have prevailed to the alarming extent which characterised the autumn and early winter months of 1895 and 1896. On the other hand, an unusual number of cases of the like condition in cattle came under observation, and as the principal facts regarding the disease are not yet so well known as they ought to be, it appears to be desirable to refer to them here at some length, although that has already been done in previous reports.

Although it is only within the last few years that the disease has been recognised and received a special name, it probably has no claim to be considered a new complaint. As a special disease of cattle, it has probably escaped notice, because it has generally been confounded with tuberculosis. It cannot be said that there ever was much justification for confounding the two diseases, for there is only a slight resemblance between them in respect of symptoms, and none whatever in the matter of lesions.

The symptoms of parasitic gastritis in cattle may be adequately described within very short compass. The earliest symptom is an appearance of unthriftiness, which soon marks the animal out from the other members of the herd. In Devons and other animals with a red coat, the hair is noticed to have acquired a yellowish tinge, especially along the back. The unthriftiness increases gradually, until it has passed into a condition of positive emaciation. Coincidentally with the loss of condition, or soon afterwards, diarrhoea sets in, and unless checked by medicines this becomes more and more profuse, and persists till death. A fact of great importance for diagnosis is that the diarrhoea and emaciation are not accompanied by any failure of the appetite, which may even be voracious until near the end. It is also important to note that there is no disturbance of the circulation or respiration, and that the temperature is normal. Occasionally there may be a little dropsy of the dewlap and beneath the throat, but as a rule there is none. Eventually the animal becomes so weak that it is unable to rise, and it is then usually killed. That is generally from three to six months after the first symptoms are noticed.

A layman may be tempted to say that the foregoing description accords pretty well with what is popularly understood by the term "consumption," and that, contrary to what has been said above, there appears to be ample excuse for confounding the disease with tuberculosis. It is, however, nearly always easy to distinguish between the two diseases, even while the animal is still alive, and the two points that are of main importance in this connection are the following. In the first place, diarrhoea is a constant symptom in parasitic gastritis, and it is absent in the great majority of cases of tuberculosis. Secondly, when tuberculous disease has advanced to such a degree as to induce marked emaciation, and to set up diarrhoea, cough and other signs of chest trouble are almost invariably present.

What has just been said refers to the means by which one may with only a small chance of error diagnose this disease while the animal is still alive, and a word or two must now be said regarding the diagnosis after death. When a *post-mortem* examination is available it is, or at least it ought to be, impossible for anyone to mistake parasitic gastritis for tuberculosis. When an animal has actually died from the latter disease, or

been killed when near the point of death, the most careless observer could not overlook the presence of the tubercles and other structural alterations that are characteristic of that disease, for no animal is ever killed by tuberculosis or even made visibly ill by it while the disease is still of microscopic extent. In advanced cases the lesions are always so conspicuous that they force themselves on the attention as soon as the body is opened.

On the other hand, in a case of parasitic gastritis, unless the person making the *post-mortem* examination knows precisely what organ to examine, and what to expect there, he is very likely to be much puzzled, for, apart from the thinness of the blood and the absence of fat throughout the body, all the organs with one exception may appear to be perfectly normal, and even this exceptional organ might pass for healthy to a casual observation. The constant seat of disease is the fourth or true digestive stomach, and the cause of the disease is the presence in this organ of parasitic worms, which although not exactly microscopic, are so small that the sharpest vision is unable to detect them in the stomach contents. The lining membrane of the stomach rarely presents any very marked signs of acute inflammation. There may be a little congestion at some places, but frequently the mucous membrane is abnormally pale, and in the worst cases its folds are thickened to a remarkable degree by a thin, clear, watery liquid, which drains away from them when they are cut across. The worms are readily detected when the stomach contents or scrapings from the mucous membrane are examined under the low power of the microscope. Two different species of worms appear to be responsible for this trouble, and in a considerable number of cases both species are found to be represented in the same stomach. The smaller species is named the *strongylus gracilis*, and the larger species the *strongylus convolutus*. Worms of both sexes and eggs are found in the stomach, but the latter pass into the intestine, and either they or the embryos hatched from them are discharged with the excrement. In this way the pasture on which such scouring animals are kept becomes contaminated, and fresh cases of the disease are produced.

That the disease is far from rare may be inferred from the fact that during the last twelve months animals so affected from seven different farms were admitted to the College Infirmary for *post-mortem* examination or experimental treatment, and in a number of other cases the disease was diagnosed by the examination of stomachs sent to the laboratory.

In previous reports attention has been called to the intractable character of the disease, and it must be admitted that the recent attempts to effect a cure have been no more successful than the earlier ones. During the past years animals have been treated with large doses of thymol and lysol, but both these substances appear to be powerless so effect a cure when emaciation has set in before the treatment is begun. The following may be cited as an example of the futility of treatment, at least in advanced cases. On 3rd November last, half an ounce each of thymol and chloroform, dissolved in equal parts of warm methylated spirits and water, was administered by means of the stomach pump to a heifer affected with the disease. Ten days later, one ounce of thymol similarly dissolved was administered in the same way, but in spite of this and a liberal diet, the heifer steadily lost condition, and, owing to inability to rise, it had to be killed on 10th December. The *post-mortem* examination confirmed the diagnosis, numerous live worms being found in scrapings from the mucous membrane of the fourth stomach. At the present time, therefore, the most economic plan would appear to be to dispose of affected animals to the butcher as soon as the condition is recognised. It is important to remember that if treatment is adopted the diseased animal ought to be kept indoors, and the manure ought to be destroyed.

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EQUINE MALARIA AND ITS SEQUELÆ.

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THE disease generally known in our sub-continent by the name of "biliary fever" of horses I have called "equine malaria." In thus indicating the nature of the disease we are at the same time able to allocate it to its proper class, for biliary fever is a disease of the blood, or, to be more accurate, a disease of the red corpuscles, these being infected by the specific parasite. In previous publications I opined that this parasite belonged to the genus *plasmodium* or *hæmamoeba*, the genus to which the parasite of human malaria belongs. I came to this conclusion on account of the multiform appearances of the endoglobular parasite in stained preparations. Since then, however, better staining methods have enabled me to come to the conclusion that the parasite in question belongs not to the genus *hæmamoeba*, but to the genus *pyroplasma*, the parasite being closely related to the *pyroplasma bigeminum* (*pyrosoma* or *apiosoma bigeminum*) and *pyroplasma canis*.

These parasites are both known in South Africa; the former causes the redwater of cattle, the latter malarial biliary fever of dogs. Laveran, to whom I sent specimens of blood taken from horses, gave this parasite the name *pyroplasma equi*, and this is the name by which I shall in future designate it.

By using Laveran's method of staining the pyroplasmodic nature of the parasite is easily seen, whereas by staining with the ordinary basic aniline dyes the parasite shows a variety of forms, such as are

commonly found in the æstivo-autumnal fever in man. They differ, however, from the parasite of human malaria (1) in not producing any pigment, and (2) in the method of reproduction.

It is convenient to retain the term malarial fever, inasmuch as the similar diseases of other domesticated animals are termed malaria by the different authors. For instance, the Italians Celli and Santori, Lignières of Argentina, and Nicolle of Constantinople, call redwater "bovine malaria," and Hutcheon of Cape Town gives the name of canine malaria to the similar malady of dogs.

Before describing the pyroplasma equi I wish to describe briefly the well-known pyroplasma bigeminum (*alias* pyrosoma bigeminum) of redwater.

We know at the present time two different forms of pyroplasma in South Africa—the small rod-shaped or bacillary form, which produces the atypical virulent redwater; and the large oval and round form, which is found in the common typical redwater. Between these two extremes every variety of intermediate form, both in shape and size, is found.

It is especially the large form I am here discussing, as commonly found in the typical forms of Texas fever all over the world.

The individual hæmatozoon is usually pear-shaped, and it was called the pyroplasma bigeminum from the parasite so frequently occurring in pairs.

A full-grown pyroplasma is often as long as the diameter of a red disc, but usually about half to two-thirds that length. The large forms are not always pyriform, but very often spherical, oval, or spindle-shaped. When stained by Laveran's method a distinct karyosome is found, which takes a deep red colour, surrounded by a clear zone which lies in a slight bluish mass of protoplasm. The karyosome is always found at the edge of the pyroplasma. The spherical forms are generally found in the internal organs, especially kidneys and heart muscle.

The pyroplasma equi is also represented by a similar variety of forms to that seen in redwater. I found stave-like, leaf-like, club-shaped, oval, pyriform, and round-shaped parasites.

Round and ring forms are very common, and the parasites are found equally distributed in the different organs. When the blood of horses suffering from the disease in its early stage is taken and defibrinated, and kept for some time before cover-glass preparations are made, nearly all the parasites appear spherical. A similar phenomenon can also be observed with pyrosoma bigeminum.

The diameter of pyrosoma equi is from $1.5\ \mu$ to $2.5\ \mu$, and the largest form fills about one-fourth to one-third of the red disc.

The smallest forms are motile, inasmuch as by careful observation they can be seen to change their place within the corpuscle. The larger forms are non-motile.

In unstained fresh preparations the parasite can easily be detected by its different refraction, thus standing out in clear outline.

I repeatedly found that fresh unstained parasites appear commonly as the round form; this is, in my opinion, an indication that its real form is the sphere, and that the non-spherical forms above described are probably dependent upon the fixation and staining of the blood film or other influences.

In the non-stained hæmatozoon no structure whatever can be detected. When stained according to Laveran's method, however, a deeply red stained body, the karyosome, can be observed in the slightly blue tinged protoplasm. This karyosome also shows different shapes; it is never in the centre, but always close to the edge. Very often around this nucleus a clear unstained or only slightly stained zone can be detected.

Reproductive forms may be found in unstained preparations, but they are more frequently seen in the stained preparations. When ordinary aniline dyes are used, a form somewhat resembling a rosette, with four leaves, can be seen connected to a centre by very fine threads. When stained by Laveran's method these forms show a karyosome in every leaf. It must be stated here that the rosette form is better shown by using the ordinary single dye than when the double stain is used, where all forms of parasites are more universally spherical. Leaf-like bodies into which the parasite splits are preceded by forms where, in a quadrangular or round mass of generally bluish-stained protoplasm, four distinct karyosomates can be observed lying crossways, thus indicating the subsequent fission with the four leaves. Besides the fission into four parts, a division into two can also be observed, but in my experience the rosette form is the most commonly found. Reproduction takes place in the circulating blood as well as in the organs, but is very frequently met with in the spleen, where the parasites may also be seen outside the red blood corpuscles. In the method of reproduction the pyroplasma equi differs somewhat from the pyroplasma bigeminum; in the latter parasite the division into two is the common way of reproduction, whereas the division into four is the rarer method.

Lignières states in a paper upon "Tristezza" that he succeeded in cultivating the pyroplasma bovis outside the animal body in cattle serum containing hæmoglobin. For this purpose he takes the blood of cattle suffering from the disease which shows abundant parasites, and distributes the blood into test tubes; he finds that in some of them a veritable proliferation takes place, which continues when fresh serum containing hæmoglobin is inoculated with the serum showing the proliferating forms.

I have on several occasions kept blood from a horse suffering from equine malaria, and which contained the pyroplasma, in test tubes at the temperature of the ice box, of the room, and of the incubator. I have found that the parasites disappear from the blood kept in the incubator as soon as the corpuscles lose their hæmoglobin; that they keep their colorability longer at the temperature of the room than at that of the incubator; and that even after twelve days specimens kept in the ice box showed the pyroplasmata as distinctly as fresh ones.

It was seen, however, that the variety of shape of the parasites had disappeared, or was only exceptionally seen, and that nearly all appeared spherical. It was noticeable that the parasites were commonly seen at the edge of the red discs, but never or very exceptionally outside the blood disc. Some of the red discs which contained the pyroplasma did not take the stain in the immediate neighbourhood of the parasite, so that it seemed to be embedded in a white zone. It is possible that this phenomenon indicates a

destruction of the hæmoglobin by some excretions of the parasite. As already mentioned, pyroplasmata were seen exceptionally in the surrounding serum. In the red corpuscles kept in the ice box, however, the reproductive form could be observed as long as four days after the removal of the blood from the horse, and the interpretation of this observation would be, that the reproduction continued even in corpuscles out of contact with the animal from which they are derived. The possibility of staining by Laveran's method both karyosoma and plasma distinctly after twelve days in the ice box, and not so after the same length of time in the incubator, indicates that the pyroplasma in the former case must have been still alive, the colorability of a nucleus being an indication of its being alive. Although transplantations into fresh serum were made, a proliferation, as mentioned by Lignières, could not be seen.

Pyroplasma equi is always present in equine malaria, and can be constantly found when the blood is examined early in the disease. It is, however, not constantly present during the whole course of the disease, and it is quite possible that one may diagnose a case as one of typical biliary fever and a subsequent microscopical investigation may fail to reveal the presence of any intra-corpuscular parasite; yet notwithstanding the total absence of the parasite the horse may die. I shall refer to this when I deal with the sequelæ.

The administration of quinine and chloride of ammonium results in a rapid disappearance of the pyroplasma, but I have on the other hand also seen a similar rapid disappearance in horses which were not treated at all, because, from the general condition of the horse no treatment was necessary. In all these instances the clinical symptoms of malaria were present for some time after the parasite had apparently disappeared.

Upon one occasion I have seen what appeared to be a karyosoma persist for a considerable period, and I have little doubt that careful observation will show that the phenomenon will not be altogether unusual. The following is the account of the case:—

5th March 1902. Brown English hackney mare, belonging to Dr T., shows symptoms of biliary fever; temperature 105° F., seedy, yellow conjunctival mucous membranes of the eye, urine yellowish, and dung of the same colour. Treated with quinine. Blood microscopically examined shows the presence of numerous parasites; all the previously described forms are present, especially the multiplication forms.

6th March 1902. Same symptoms as the day before, no improvement. Quinine is given three times a day, and chloride of ammonium in a half ounce dose.

7th March 1902. Same symptoms, but the animal shows marked improvement: treatment continued. Pyroplasmata appear to be less numerous in the blood.

8th March 1902. Symptoms of jaundice are still very marked, but the mare has improved, feeds very well, and is very lively. She stales frequently and abundantly, the urine being of a yellow colour. Parasites are very rarely met with in the blood; at the commencement from five to twelve parasites could be detected in each microscopic field, now as many fields must be searched in order to discover one parasite.

The mare recovered, but the blood was examined daily up to 24th March 1902 (*i.e.* for nineteen days). During the whole of this period there was constantly present a round body in a red corpuscle, which, when stained by Laveran's method, was of a deep red colour. This reaction to the stain shows that the body is of the nature of a karyosome. No protoplasmic tissue could be traced, not even after using different stains. This chromatic body was of the size of the karyosoma in the full developed parasite previously described; a further peculiarity was that at the beginning it could only be found in the so-called macrocytes, or corpuscles of about double the size of an ordinary erythrocyte: and it was not until towards the end of the observation that normal-size blood discs containing these bodies could be observed. It may be stated here that in this mare's blood both megaloblasts (*viz.*, large corpuscles with a nucleus) and macrocytes were repeatedly found, indicating the acute anaemia which was caused by the invasion of pyroplasma. But the macrocytes disappeared rapidly after recovery. More than one body in one corpuscle was never seen: this fact would exclude the possibility of its being simply a basic alteration of the hæmoglobin.

I consider the chromatic body to be one of the forms in the development of the parasite, and have no hesitation in thinking that it represents a latent or dormant form. For this reason I have eschewed the term spore.

Inoculability of the Disease.—In order to prove conclusively that a given micro-organism is the *causa causans* of a disease which is characterised by its presence, the inoculation of it into other animals should produce the same disease again, provided the animal operated upon is not immune against the disease. I repeatedly injected horses with the blood of equine malaria which contained the pyroplasma, but up to the present I have been unable to transmit the disease in this way from one animal to another. At first I injected small quantities of blood subcutaneously, later intravenously into the jugular vein; then, seeing that all these efforts failed, simply connected the jugular vein of a healthy horse by means of indiarubber tubing to the vein of a sick one, and let the blood run into the former for a period of two minutes. The amount of blood thus infused I calculated to be about one litre. Even this procedure failed to reproduce the disease. This failure is somewhat disheartening, seeing that the other diseases caused by pyroplasmata, namely pyroplasma bigeminum and pyroplasma canis, can be transmitted by injection of blood which contain them. But it must be borne in mind that these two diseases of cattle and dogs, respectively, can only be transferred in this way to *susceptible* specimens of their species. For instance, I have repeatedly tried to produce redwater by injecting virulent blood into oxen and calves born in and around Pretoria (a badly tick-infected area), and I have never succeeded in producing the disease, whereas freshly imported cattle from the Cape peninsula easily contract the disease. The same fact has been repeatedly proved for cattle born in other redwater countries.

This analogy points to the fact that my experimental horses must have had an immunity against biliary fever; in fact, all the animals experimented upon were South African veldt horses, and it is not improbable that they had either an acquired immunity, from a

previous attack of the disease, or a natural immunity. In order to show that a horse can be infected by the injection of infected blood, freshly imported horses will have to be injected. The failure to infect South African horses with equine malaria—by analogy with the other pyroplasmic diseases—would point to a high degree of immunity in the locally bred horses.

The question arises, how does a horse become infected by the pyroplasma equi?

The conclusion has again to be drawn from the analogy of the similar diseases in cattle and dogs. It has been proved that redwater is carried from a sick animal to a healthy animal by ticks. In America it was found that the tick *rhhipicephalus annulatus* is the propagator, and in Australia the tick *rhhipicephalus australis*. In these countries the disease has always followed the ticks, and the conclusion drawn therefrom could be proved by experiments. In South Africa the tick was known before redwater, and it only became infected after the importation of cattle suffering from the disease. The farmer, therefore, would not allow that the ticks had anything to do with the disease, and generally ridiculed the idea. It has, however, now been proved that the common blue tick of South Africa, *rhhipicephalus decoloratus* (Koch), is really the propagator of redwater. Recently the entomologist of Cape Colony, Lawnsbury, has proved that malignant malaria in dogs is due to the tick *hæmophysalis leachi* (Audin). The experiments with ticks show that the female tick transmits the infection into the eggs, from which it passes into the larvæ and shows in the pupa. Larvæ and pupæ can feed on healthy dogs without transmitting the disease, but as soon as they moult the imago or adult tick transmits the disease.

We have to conclude therefrom that in the different stages of the tick the pyroplasma undergoes changes of form in the process of reproduction.

It has been shown that the resemblance between the different forms of pyroplasmic diseases is very great; for two of them it has been proved beyond doubt that they are transmitted by ticks; the conclusion is, therefore, that the propagation of the third disease, caused by pyroplasmata, viz., malarial fever in horses, is also due to the same agent. It should be possible to prove this by experiment, but, as I have said, I have up to now been unable to do so, on account of the want of freshly imported horses. Observations made during the recent campaign go to support the idea. We noticed that biliary fever was nearly exclusively confined to imported horses; nor did it seem to matter from what country the horses were derived, for, so far as I can find from the literature, malaria in horses is unknown in all the countries from which they were derived. I had the opportunity on several occasions of observing that imported English horses which were in Pretoria, and which were kept under circumstances similar to those at home, viz., stabled and properly groomed, fell sick with biliary fever when they were turned out to graze and allowed to become tick-infected. In two instances the observation was so striking that it could be quoted as an experiment. The ticks commonly found, viz., the blue ticks (*rhhipicephalus decoloratus*), are very likely also responsible for the propagation. This arachnid has already been shown to convey redwater. The question, therefore,

whether these two diseases are identical naturally arises. The pathological appearances are also very similar, and the fact that hæmoglobinuria is not seen in horses can be explained in the same way as those cases of redwater in which the discolouration of the urine is absent, namely, to a greater resistance of the red corpuscles. Although I do not believe that the two diseases are identical, I inoculated horses several times with blood from cattle suffering from redwater without ever noticing any subsequent disease. The geographical distribution of horse malaria and redwater, moreover, points out that they do not result from the same cause. According to Hutcheon, biliary fever can be observed in the Cape Peninsula, a territory which, as far as I am aware, is not yet infected with redwater.

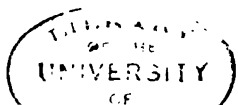
I also tried injecting blood from horses suffering with malaria into cattle, but without effect; and I have at different times tried to infect the ordinary experimental animals, such as rabbits, dogs, and guinea-pigs, but I failed to produce any reaction. The conclusion may therefore be drawn that malarial fever in horses is a specific disease peculiar to the species *equus caballus*. The injection of the blood of horses suffering from malaria into the peritoneal cavity of guinea-pigs was without effect, whereas the blood from cattle containing *pyrosoma bovis* had an immediate effect on these animals, producing symptoms of spasm, cramps, and even sudden collapse.

Before concluding these notes on *pyroplasma equi*, I wish again to refer to the immunity of horses against this disease. I have no proof that malarial fever leaves any immunity whatever, but we must assume that such is the case. The immunity of South African veldt horses has, however, to be explained differently, and not by assuming that they have really passed through the disease. The analogy to redwater again gives the clue to the solution. We know that calves possess a great resistance against redwater, and when they are born in a redwater country we know that they acquire immunity which lasts for a considerable time, probably for life. This is due to the constant infection during the early life, when they are already very resistant. The same will hold good for biliary fever. The foal, whilst running with its mother in the fields, becomes tick infected, and whilst highly resistant on account of its youth is gradually vaccinated. When full grown it is thus immunised against the disease.

Sequelæ of Malarial Fever.

From the above notes it can be seen that I consider that the *pyroplasma equi* is the *causa causans* of biliary fever. I have stated that it is to be found in the early stages that it is not found in healthy horses or in horses suffering from other diseases; and I have demonstrated how quickly it leaves the animal body (the resistant forms excepted) when the horse is recovering. In some horses which do not recover I have noticed that the *pyroplasma* is present in the blood and internal organs up to the time of death; in others, however, it is absent.

It is exceedingly rare to find that only the *pyroplasma* is found in a horse suffering from and dying of biliary fever. In nearly every such case I found a bacterium which was present sometimes in



the blood, and always in the spleen, and which very often could be traced *intra vitam* by the agglutinating properties of the serum of such animals as were infected by it.

Before entering, however, into the life history and the characteristics of this bacterium, it will be advisable to quote illustrative cases.

(1). 14th July 1900. Argentine gelding, six years old. This horse was observed to be ill, temperature $40\cdot5^{\circ}$ C. The pulse was very rapid and feeble. The respiration was accelerated, and finally "pumping." The mucous membranes were spotted with red. On auscultation crepitation could be distinctly heard. The animal died during the night.

A *post-mortem* was made early in the morning. The lungs were in a state of inspiration. The interlobular tissue was œdematous at the apices and roots of the lungs and at the base of the heart. The bronchi were filled with white foam. The blood was coagulated. In the right ventricle a solid clot was found, and there was a smaller one in the left one. A few ecchymoses were found in the endocardium. The spleen was much enlarged; the pulp, however, was fairly solid. The stomach was normal. In the small intestines a few red patches were seen and injection of the blood vessels. The liver was jaundiced. A general slight jaundiced condition of all the organs was present.

The microscopic examination showed the absence of endoglobular parasites; in smear preparations from the spleen the bacterium could be found, and cultivations made from the blood and the spleen on agar-agar proved it to be present in pure culture. From the state of the lungs, some doubts were entertained whether this was not a case of "dunkop" (horse-sickness), although the season in which it occurred was against it. Some blood was therefore injected into a horse; horse-sickness did not follow, however; and, as the experimental horse died from a subsequent inoculation from horse-sickness, it was proved that the lesions in the lungs of the former horse had nothing to do with this disease.

(2). 2nd August 1900. Chestnut gelding, seven years old. Arrived the day before into the sick lines with symptoms of "pulmonary œdema." The respiration was very much accelerated and the nostrils wide open. Crepitation could distinctly be heard in both lungs. The pulse was imperceptible. The heart beats exceeded 80 per minute. The conjunctival mucous membrane showed ecchymoses. Temperature $38\cdot5^{\circ}$ C. There was a serous discharge from the right nostril. Blood was taken both for examination and culture. The horse died during the night.

Post-mortem early in the morning. The lungs were in the extreme of inspiration. The bronchi were full of foam. On the inner surface of the lungs on both sides it was seen that the pleuræ were infiltrated with yellowish lymph. The interlobular tissue at the edges of the apex were distended with the same lymph. The mediastinal glands and those of the lungs were enlarged and gorged with serous fluid. The pericardial sac contained serous fluid. The heart was one mass of petechiæ; the heart muscle between the hæmorrhages was of a greyish colour, and appeared as if it had been boiled. In both ventricles were yellow clots of coagulated blood. The liver was enlarged, of a dark yellow colour, and had a granular appearance. The bile

ducts were gorged with greenish-looking bile. The spleen was very much enlarged, the pulp fairly hard; the lymphatic glands were enlarged and hæmorrhagic. The kidneys were normal. There was no pathological change in the stomach or intestines.

The microscopic examination of the blood and spleen showed the absence of endoglobular parasites.

No cultures were made from the spleen, but from the blood the bacterium was found in pure culture.

There was a doubt whether the case was not one of horse-sickness. Two experimental horses were therefore injected with blood, but horse-sickness did not follow the injection.

(3.) 26th August 1900. English mare. This mare had been some time in the sick lines, and had been treated for a wound on the left fetlock. Beyond the wound nothing was noticed to be wrong with her until the morning of the 25th August, when she was found on the ground, with symptoms of spasm, which returned periodically. The head and legs were alternately drawn up under her belly and suddenly extended. A peculiar rolling of the eyes could be noticed, thereupon the horse moved its legs as if to walk, finally it became comatose and died during the night.

Post-mortem early next morning. The mare was in pretty good condition. With the exception of a very large spleen, nothing pathological seen with the naked eye. From both ventricles long yellow clots of coagulated blood were drawn.

Microscopic examination of smear preparations from the spleen showed the presence of the bacterium, which was cultivated in pure state on agar-agar.

(4.) 15th September 1900. English gelding, eleven years. This horse arrived the previous evening in the sick lines and died shortly afterwards.

The *post-mortem* was made early on the morning of the 15th. A general jaundiced condition of the flesh and all internal organs could be noticed. The spleen was enormously enlarged; it reached backwards nearly as far as the ileum. The pulp of the spleen was soft and black. The liver was also enlarged and yellow, the bile ducts filled with bile. The kidneys were anæmic. The lymphatic glands of these mentioned organs were enlarged with blood infiltrations. The urinary bladder contained brownish-looking urine. The stomach and intestines were empty; the mucous membrane of the stomach was very pale; the mucous membrane of the small intestine looked like eel-skin. The lungs were in inspiration, contained much blood, and were oedematous. The heart was found in diastole, and was filled with black blood clot. Preparations of the spleen showed the presence of the bacterium.

(5.) 3rd October 1900. Chestnut gelding, eight years old. Died to-day with convulsions.

Post-mortem about three hours later. Condition good. The spleen was about three times its normal size, the pulp was very soft; its lymphatic glands enlarged and infiltrated with blood. The liver was also enlarged and very yellow. The kidneys were normal. The stomach was normal; the mucous membrane of the small intestine was of slate colour, and that of the colon was deep black in parts. The lungs showed emphysematous spots, but were otherwise normal.

The heart was in diastole, and both ventricles contained well coagulated clots. The myocardium looked as if it had been boiled.

Microscopic examination of smear preparations showed the absence of endoglobular parasites, and the presence of the bacterium, which grew in pure culture on agar-agar.

(6.) 8th October 1900. Brown pony gelding. Arrived in the sick lines on the previous evening with a temperature of 103·6° F. and died soon afterwards.

Post-mortem was made early on the 8th. The skinned cadaver and all internal organs showed a generally jaundiced condition. The spleen was enormously enlarged, its weight being 8·5 lbs., and the pulp was soft. The liver was also enlarged, very yellow, and friable. Both kidneys were enlarged and very pale. The lymphatic glands of these three organs and of the stomach were enlarged and infiltrated with blood. The mucosa of ileum, jejunum, and colon was of a slate colour; the pericardial sac was filled with brownish-yellow fluid. The myocardium looked as if it had been boiled. Subendocardial petechiæ were very marked in the left ventricle. Both ventricles contained yellow clots.

In smear preparations of the spleen no endoglobular parasites could be seen under the microscope, but cultivations made on agar-agar proved the presence of the bacterium in pure culture.

(7.) 24th October 1900. English chestnut mare, nine years old. She showed all the symptoms of biliary fever, and the microscopical examination of the blood proved the presence of pyroplasma equi. Symptoms of pneumonia were also present, and the neck was swollen. The horse died in the night.

Post-mortem was made about nine hours later. There was a generally jaundiced condition of the flesh and all the internal organs. The spleen weighed 9 lbs. 4 ozs.; the pulp was very soft. The liver was enlarged and yellow. The kidneys very pale. The pericardium was covered with a yellowish dry mass. In the pleural cavity serous fluid was found. The lungs around the heart were in a state of engorgement, and surrounded by œdematous infiltration. The pleura at this particular point was thickened and injected. Foam was present in the trachea. In both ventricles were yellow clots. The myocardium appeared as if it had been boiled, and was very friable. All the lymphatic glands were enlarged and infiltrated with hæmorrhages.

Smear preparations from the spleen showed the presence of the pyroplasma equi, and the bacterium was cultivated in pure culture.

(8.) 24th October 1900. English chestnut mare, thirteen years old. Died very suddenly in the night.

Post-mortem early in the morning. The mare was in pretty good condition. Rigor mortis was present. The spleen was very much enlarged, and weighed 8 lbs. 2 ozs. The mucosa of the stomach was of a slate colour. The small intestines were slightly congested. The liver was very large and congested. In the left ventricle, some white fibrous streaks. The remainder of the organs were normal.

The bacteria were found by culture in a pure state.

(9.) 1st November 1900. English mare, seven years old. On 24th October 1900 the diagnosis of biliary fever was made both clinically and by microscopic examination. The horse was treated with quinine, and showed improvement for some days. On the 28th

October no more pyroplasmata could be found in the red corpuscles, and the animal had a very good appetite. On the 29th October, however, a change for the worse took place; the evening temperature rose to 40° C., the hind legs began to swell, and the pulse was very feeble. The animal died during the morning of the 1st November.

Post-mortem was made about one-and-a-half hours after death. The cadaver was in a poor condition. The flesh had a brownish tint. All the fasciæ and serous membranes were yellow. The blood was very well coagulated. The peritoneum contained yellow fluid. The spleen was slightly enlarged, and weighed 2 lbs. 4 ozs.; the pulp was of a brownish colour and rather hard. The lymphatic glands of the spleen were enlarged and infiltrated with hæmorrhages. The liver was jaundiced and contained much blood. Both kidneys were enlarged and surrounded by œdematous capsules; the cortex seemed to be broader than usual and yellow. The subrenal glands were swollen. The stomach and intestines were empty. The mucous membrane of the colon was slightly reddened. The lungs were in inspiration and emphysematous at the edges. In the middle of the right lobe there was a pneumonic patch. The left ventricle contained subendocardial hæmorrhages.

Microscopical examination of smear preparations made from the spleen showed the absence of endoglobular parasites and the presence of the bacterium.

This bacterium also grew in cultures made from blood taken on the 27th October, on which day a striking decrease of endoglobular parasites could be noticed.

(10.) 2nd November 1900. English gelding, eight years old. This horse arrived in the sick lines the previous day with a temperature of 105·4° F. The respiration was accelerated; auscultation revealed increased vesicular sound on both sides. The pulse was 60. Conjunctiva bulbis was yellowish, with hæmorrhagic spots. The lower part of the head was swollen, and the head was held in a hanging position. In the evening of the same day the thermometer registered 104° F., the swelling increased. The horse became comatose and died in the morning of 2nd November 1900.

The *post-mortem* was made one-and-a-half-hours after death. Cadaver was in a poor condition. The flesh and all the internal organs were jaundiced. The blood was only partially coagulated. In cutting the axillary vein a brown serum ran out. The spleen was enormously enlarged and weighed 10 lbs. The pulp was black and protruded when a cut was made into the capsule. The liver was also enlarged, of a brownish-black colour, and strongly congested. The kidneys were enlarged and also of brownish-yellow colour; the cortex was pale. All the lymphatic glands were enlarged and infiltrated with blood. The pericardium contained serous fluid. The heart was flabby, and there were subendocardial hæmorrhages in the left ventricle. The lungs were emphysematous. The mucous membrane of stomach and intestines was brownish-yellow; no hæmorrhages were present, but there was a layer of tough mucus.

Microscopic examination of blood from the heart and of smear preparations from the spleen showed the absence of the pyroplasma equi. The bacterium, however, could be found in cultures made from the blood, spleen, kidney, and liver.

(11.) 4th January 1901. Horse 1050. English gelding, dun-coloured, nine years old. This animal arrived four days previously into the sick lines with high fever. The mucous membranes were yellow, and in the course of the succeeding days became spotted with hæmorrhages. The pulse was very bad from the beginning. The condition changed rapidly for the worse. Swellings appeared around the mouth, and a yellowish diarrhœa set in. The horse died in the night of the 3rd January.

The *post-mortem* was made early in the morning of the 4th. The cadaver was in poor condition. Rigor mortis was present. The flesh was sepia-colour. The spleen weighed 5 lbs; its lymphatic glands were enlarged, œdematous, and partially infiltrated with blood. The liver was enlarged and yellowish-brown, of nutmeg character; all the lymphatic glands were enlarged and œdematous. The kidneys were enlarged and very pale. The mucous membrane of the small intestines showed slate colour. The lungs were in inspiration, and at both apices were capsulated abscesses. The heart muscle was also of a sepia-colour. In the left ventricle were numerous subendocardial hæmorrhages.

Microscopic examination showed the absence of the pyroplasma equi in smear preparations of the spleen. The bacterium was obtained therefrom in pure culture.

(12.) 5th January 1901. Horse 17. Gelding, eight years old. This animal was for some time in the sick lines suffering from laminitis. It was seen to be very ill. The respirations were accelerated and pumping, the pulse imperceptible, temperature 41° C., mucous membrane of the eye reddened. The horse was obviously dying; it dropped and showed convulsions of the neck and legs, and it was therefore killed.

Post-mortem about one hour later. The condition was good. The spleen was enlarged and weighed 5 lbs; the pulp was soft. The kidneys were enlarged. The malpighian bodies were very distinct. The lymphatic glands of the kidneys were very much enlarged and infiltrated with hæmorrhages. The liver was greatly congested and of a dark brown colour. The mucous membrane of the small intestines was reddened in patches and injected. The lungs were in semi-inspiration, and spotted with small hæmorrhages. The heart seemed to be normal. The blood was not properly coagulated and was black.

From the spleen the bacterium was obtained in pure culture.

(13.) 19th January 1901. English mare, nine years old. This horse was for some considerable time in the sick lines, and was in very poor condition. A definite diagnosis was, however, not possible. It became lame all of a sudden on the near fore leg, and at the same time an acute iritis was observed. The animal was killed on the 19th January 1901, as *exitus letalis* was anticipated.

Post-mortem immediately after death. In the lungs numerous small encapsulated abscesses and emphysematous spots could be observed. The endocardium of the left ventricle was thickened and white. Slight tumour of the spleen was present; its weight was 4 lbs. The kidneys were dark red and markedly congested; there was some difficulty in removing the capsule. In the medulla white streaks extending into the cortex could be seen. The liver was enlarged and

friable. The stomach and bowels were normal. Under the coronary band an abscess was found. In the frontal chamber of the eye was a yellow coagulum.

Cultures made from the spleen gave the bacterium in pure state.

(14.) 12th March 1901. Horse 34. Gray mare, ten years old. This horse arrived in the sick lines on 2nd March, when both clinically and microscopically bilious fever was diagnosed. It was treated at once with quinine, and the pyroplasma soon disappeared. It did not, however, improve, but a general marasmus supervened. The horse died in the night of the 12th March 1901.

Post-mortem was made early in the morning. The cadaver was thin. The flesh was of a yellowish colour. The spleen was enlarged and weighed 4 lbs.; the pulp was soft and of a brownish-yellow colour. The liver was enlarged and nutmeg in character, very fatty and friable. The kidneys were enlarged and pale, not unlike the colour of burnt sienna. The stomach and bowels were normal. The lungs were œdematous. On the surface of the left lobe was a circumscribed pleuritis fibrosa and fibrinosa, underneath a grey pneumonic patch. In the left ventricle were subendocardial hæmorrhages. The pericardium was injected.

In smear preparations from the spleen only bacteria were found, which were obtained in pure culture.

(15.) 21st January 1902. Australian mare, eight years old; in very poor condition. The mucous membranes were very pale and showed petechiæ. The horse died during the night of 20th January 1902.

The *post-mortem* was made early next morning. A general anæmia of all organs was very well marked. The endocardium was full of hæmorrhages. The spleen was enormously enlarged, and the pulp soft.

Smear preparations from the spleen showed the absence of the pyroplasma. The bacterium was obtained in pure culture.

(16.) 12th February 1902. English mare, six years old. This horse showed clinically all the symptoms of biliary fever. Microscopical examinations proved the presence of the pyroplasma equi. The horse was treated with quinine and stimulants, and under this treatment it improved. A change for the worse took place on the 19th, and the horse died on the morning of the 20th February 1902. During the whole time the pyroplasma equi was present in the blood, but was less numerous at the end of the disease than at the beginning.

The *post-mortem* was made shortly after death. The blood was well coagulated. The flesh was of a brownish-yellow colour. The spleen weighed 7 lbs.; its pulp was soft, and the lymphatic glands were enlarged and infiltrated with blood. The liver was enlarged, of a reddish-brown colour, and of nutmeg character. The kidneys were pale. In the stomach and bowels nothing pathological could be found. The lungs were in half inspiration and were slightly œdematous. The heart muscle appeared as if it had been boiled. There were subendocardial hæmorrhages in the left ventricle; all the lymphatic glands were generally enlarged.

In smear preparations of the spleen the pyroplasma equi was present; the bacterium was obtained in pure culture.

(17.) 13th March 1902. Chestnut pony gelding. This horse

arrived some time previously from Heidelberg, where it had been suffering from bilious fever five weeks before this second disease. For a few days it had high fever; it was impossible, however, to arrive at a diagnosis. The mucous membranes of the eye were very pale and spotted with hæmorrhages.

The animal died during the day of 13th March 1902, and a *post-mortem* was immediately made. The blood was very thin and of a greenish colour, containing when defibrinated a very small deposit of red corpuscles. The cadaver appeared as it had been bled to death. The spleen was enormously enlarged and weighed 13 lbs., 10 ounces; the lymphatic glands of the spleen were enlarged and infiltrated with hæmorrhages. The kidneys were œdematous, enlarged, and very pale. The liver was very friable and fatty. The myocardium was of a sepia colour, and very friable. Subendocardial hæmorrhages were present. The mucous membrane of the stomach had a few erosions which were covered with coagulated blood. The mucous membrane of the bowels was swollen and had a yellowish glass-like appearance. In the colon ecchymoses could be found on the mucous membrane. The lungs were also spotted with very fine hæmorrhages.

Microscopic examination of specimens from the different organs showed the absence of any endoglobular parasite. From the spleen the bacterium was obtained in pure culture.

(18.) 22nd March 1902. Horse. S.A.C., dun. This animal had been suffering for some time from malarial fever. A diagnosis to this effect was made by Vet. Captain Christy of the S.A.C.

Post-mortem was made about two hours after death. The cadaver was in very poor condition and anæmic. The flesh and all the organs had a generally jaundiced condition. The spleen was very large and soft. The liver was also enlarged, quite yellow, and friable. The kidneys were pale, but looked otherwise normal. In the lungs capsulated abscesses were found.

Smear preparations from the spleen showed the absence of the pyroplasma equi. Cultures showed the bacterium.

Analysis of the foregoing Cases.

These sixteen cases may be divided into four groups:—

(1.) The cases in which the pyroplasma equi was found in the blood both during life and *post-mortem* (4, 7, and 15).

(2.) The cases in which the pyroplasma equi was found *intra vitam*, but was not found *post-mortem* (8).

(3.) The cases in which no pyroplasma was found shortly before death, and none *post-mortem* (1, 2, 5, 6, 9, 10, 16, and 18), but where the characteristics of the *post-mortem* lesions left no doubt that the animals had suffered from biliary fever.

(4.) The cases in which no pyroplasma was found on *post-mortem*, and where the characteristic lesions were not so pronounced as to justify a certain diagnosis of biliary fever (3, 11, 12, 14). I consider these cases, notwithstanding the absence of the most striking symptoms of biliary fever, as due to previous biliary fever, and I base my opinion on the presence of the splenic tumour which was found in all cases, and upon the general degenerations of the internal organs. In these particular cases icterus was absent.

Description of the Bacterium.

Since the presence of the particular bacterium of which I have spoken was demonstrated in every case, although the pyroplasma equi was absent in the majority of these cases, a description of the bacterium may here be given before discussing the question of experimental pathogenesis.

Morphology.—It is a small rod, with rounded edges, resembling the so-called "coccobacillus," and is about the size of the bacillus hæmorrhagicus (chicken cholera). The size varies, however, according to the medium upon which it is grown. The bacillus may therefore appear to be of a iso-diametrical form, not unlike a coccus, and as a rod, about three to five times as long as broad. The former are usually observed when cultivated in broth. The staining is very often most marked at both poles, especially in young forms, and the bacilli in smear preparations of the spleen and blood show typical bipolar staining. The bacillus takes all the basic stains, but the best preparations are obtained with fuchsin gentian violet; methylene-blue stains very faintly. In smear preparations the best results are obtained with carbol-thionin blue. When treated by Gram's method the bacillus does not retain the stain.

Motility.—The bacillus shows distinct though not rapid motility.

Cultural Reactions.—When the spleen juice or blood is first inoculated the growth is not abundant. Several drop-like colonies appear, which gradually increase in size, and eventually become wrinkled on the surface. Subsequent sub-cultures result in abundant growth.

Broth.—This medium soon becomes turbid, and sometimes a pellicle is formed on the surface: a deposit is constantly formed, which is considerable in old cultures.

Gelatine.—It grows very slowly in stab-cultures in this medium, and never liquefies it. Along the stab a row of fine isolated colonies can be observed. On gelatine plates only very small drop-like colonies can be observed.

Agar.—On slanting agar the growth is very abundant after the first generation. It begins with a number of very fine drops, which become confluent; the resulting growth may become wrinkled when the growth does not reach the glass, or the growth may be thin and with a bluish iridescent lustre. Under all circumstances the growth is transparent.

On glycerine agar the growth is typical. The shrinking is extremely pronounced, and the layer is nearly translucent. The water of condensation in both agar and glycerine agar tubes becomes only slightly turbid. When touched with the platinum needle whole pieces of the shrunken culture can be removed. After several sub-cultures, however, this particular characteristic is lost, and the growth has an even surface and is translucent.

Grape-sugar agar, milk-sugar agar, and cane-sugar agar become broken up when inoculated. This is very pronounced in grape-sugar agar.

Milk is as a rule not coagulated. One sample, however, did so.

On potato there is a growth of a slight brownish colour, and the potato itself is subsequently stained the same colour.

On coagulated blood serum a translucent growth without any special characteristics is to be observed. In liquid serum the growth takes place only at the bottom of the test tube, and the serum never becomes turbid.

The bacillus is an obligatory aerobe.

No indol is formed.

Inoculation of the Culture into Horses.

(1.) 30th October 1900. South African pony, six years old. This animal was inoculated into the jugular vein with 18 cc. of a bouillon culture, fourteen days old, two generations from the original culture. Directly after injection most alarming symptoms were observed: accelerated respiration, colic, diarrhoea, and collapse, which ended in death after eight hours.

The *post-mortem* was made at once. The spleen weighed 2 lbs., 10 ounces. The liver was enlarged and strongly congested. The kidney was also markedly congested. The mucous membrane of the colon and cæcum had a generally reddened appearance, with still deeper red patches. The mucous membrane of the small intestines was here and there of a slate colour. The mucosa of the stomach was slightly reddened. The lungs were emphysematous. In the left ventricle there were subendocardial hæmorrhages. The bacillus was again recovered from the spleen.

(2.) 8th November 1900. Horse S. H. English mare, six years old. Injected with 5 cc. of a bouillon culture into the jugular vein. Symptoms of colic soon supervened; the respirations were accelerated, the temperature rose to 39·8° C. The horse seemed to be all right again on the following day.

20th November 1900. The animal received 20 cc. of a bouillon culture, four generations from the original, into the jugular vein. Again signs of uneasiness were noticed, and the temperature rose to 40° C, and during the next two days kept oscillating, always slightly above normal.

30th November 1900. The animal had completely recovered, and was again injected into the jugular vein with 20 cc. of a bouillon culture. Only a slight uneasiness could be observed, and the temperature never rose above 39·5° C.

12th December 1900. On this day the animal was injected with 40 cc. of a bouillon culture about one month old. The inoculation was made very carefully and at intervals. The animal, however, showed the most alarming symptoms of congestion of the lungs, and died suddenly a few minutes after the injection.

The *post-mortem* was made immediately. Hyeræmia of the lungs was present. The spleen weighed 2 lbs. 12 ounces. All the organs were normal. The bacillus could not be found in the spleen by culture.

(3.) African pony, twelve years old. This horse received on 12th March 1901 30 cc. of a culture taken from the horse which died on 27th February 1901. Directly after the injection symptoms of colic came on, and the horse died during the night.

The *post-mortem* was made about twelve hours later. The whole of the small intestine showed hæmorrhagic inflammation; the mucosa of the stomach was also reddened. Some parts of the

intestinal mucosa was of a slate colour, among other parts the whole of the cæcum. The kidneys were markedly congested, and the surrounding tissue œdematous. The subrenal glands showed hæmorrhages on section. The spleen was slightly enlarged and soft; the lymphatic glands of this organ were enlarged and infiltrated with blood. The liver was hard. The lungs were normal. The heart was in diastole. The blood was badly coagulated. In the right ventricle were a few hæmorrhages.

(4.) 19th October 1900. Horse 27, pony four years old. 20 cc. of a broth culture of the second generation were injected into the jugular vein. This was followed by very alarming symptoms, accelerated respiration, and diarrhœa. The temperature rose above 40° C, and remained so for a few days. General depression was also observed, but the horse finally recovered.

(5.) African pony. This horse was injected into the jugular vein on 18th October 1900 with a culture (third generation) four days old. 9 cc. were injected. Symptoms of a slight colic with repeated evacuation of the rectum followed, but the animal recovered.

(6.) Horse 45, a pony. Injected 22nd October 1900 with 10 cc. of a bouillon culture into the jugular vein. The culture had been first passed through a rabbit. Symptoms of colic followed, the animal lay near constantly, and the temperature rose to 40° C.

(7.) Horse 32. Six years old, grey gelding. On 30th October 1900 it was inoculated with 18 cc. of a bouillon culture, five days old. The injection was made subcutaneously. Only slight evidence of uneasiness was observed. At the site of inoculation a large swelling was formed, apparently very painful to touch. The bacillus was obtained from the swelling in pure culture.

On the 5th November 1900 this animal was injected into the jugular with a further 18 cc. of a bouillon culture, taken from a rabbit. Symptoms of uneasiness supervened. The temperature rose the next day to 40·8° C. On the morning of the 7th November the animal was found in the paddock in a comatose state, entangled in wire fence, and was therefore killed.

The *post-mortem* showed no characteristic changes. Bacteria were found in the spleen.

(8.) 11th March 1901. Horse 85, South African mare. The object of this experiment was to predispose the horse to the action of the bacterium. For this purpose blood was withdrawn, then the jugular of horse No. 34, which was suffering from malaria sequelæ, was connected with the jugular of horse 85 by india-rubber tubing, and for a few minutes the blood was allowed to run from horse 34 into horse 85. The infusion was followed by no untoward symptoms. At the same time 85 cc. of the blood of horse No. 34, collected and defibrinated the previous day, were injected subcutaneously.

13th March 1902. Horse 85 was injected subcutaneously with 30 cc. of a bouillon culture three days old. Symptoms of a very acute disease soon developed. The pulse was 76°, the temperature rose to 103·8°, the respirations to 68° and very superficial. The mucous membrane of the eye became very red. The mare ceased to feed, and became semi-comatose. The temperature for the next day kept at 104·4° in the evening, and the pulse rose to 90°. Respirations receded to 26°. The animal, however, recovered. The site of in-

oculation was marked by a swelling of the size of two hands, and was hot and painful. It did not suppurate. This horse was again injected, and after this injection suppuration and necrosis of the subcutaneous tissue supervened.

(9.) Grey gelding, fifteen years old. On the 21st March 1901 this animal was inoculated with 50 cc. of the filtered toxin of a fourteen days old bouillon culture. Filtration was effected by a Chamberland filter, and the toxin proved sterile when planted on agar. Soon after the injection the horse sweated profusely, the respiration was accelerated, and dung was repeatedly voided.

About an hour later the animal was injected with a further dose of 90 cc. of the same toxin. Similar symptoms were observed, but the perspiration on this occasion was so severe that the water dropped from the animal. The temperature rose to 39.5° C. The horse sat on his haunches, and his eyes were suffused with tears. The pulse disappeared. On the morning of 23rd March 1901 the horse was found in agony, and was therefore killed.

The *post-mortem* was made directly after death. The epicardium was dotted with hæmorrhages, and there were subendocardial hæmorrhages in the right ventricle. The atrio-ventricular valves were œdematous, but otherwise normal. No culture could be obtained from the spleen.

Experiments with Rabbits. 19th October 1900. Rabbit F.a. was injected with 5 cc. of a bouillon culture into the peritoneum.

20th October 1900. The rabbit died during the night.

At the *post-mortem* examination there was very little fluid in the peritoneal cavity. There were hæmorrhages on the rectum. Bacteria were present in the peritoneum, but only a few were found in the blood from the heart.

3rd September 1900. 4 cc. of a bouillon culture were injected. The animal died next day. There was a peritonitis fibrinosa, and bacteria were present in the peritoneal cavity and the heart blood.

Two more rabbits were injected under the skin with 1 cc. of the culture, but death did not ensue; nor did the rabbit injected with 1.5 cc. intraperitoneally die.

Pigeons. Two pigeons were injected with 2 cc. of a one-day-old culture. These birds refused to eat for the next two days, but recovered.

Rats. A white rat was injected subcutaneously, and another one intraperitoneally. Both showed symptoms of illness during the following days, but recovered.

Sheep. A young sheep was injected with 2 cc. of a bouillon culture into the jugular. It showed symptoms of illness during the following days, but recovered.

Goat. A young goat was injected with 5 cc. of a bouillon culture in the leg. She went very lame and was seedy, but recovered.

Analysis of the above Experiments.

The bacillus described above could not be identified as one of the already described ones. It is easily distinguished from the hæmorrhagic septicæmia group, otherwise called *pasteurella* by Lignières. In Lehmann and Neumann's classification of bacteria it would fall under the group of *bacterium coli*, although it differs from it by not

producing indol. In Flüggé's classification of micro-organisms it would fall into the hæmorrhagic septicæmia group, to which belong motile bacilli which do not exactly cause septicæmia, but which are found in the tissues herded together in masses, resembling in this particular the typhoid bacillus. The bacillus in question shows a tendency to form colonies in the spleen; smear preparations from the spleen show the bacilli always grouped together. This, and the fact that it is constantly found in the spleen, makes the resemblance to the bacillus typhi abdominalis more marked.

It has been demonstrated that the bacillus is only slightly virulent for the smaller laboratory animals, and the injection of such large quantities of culture produces death rather by toxæmia than by the multiplication of the bacteria.

The bacillus is, however, decidedly pathogenic for horses. Death after injection into the jugular is, in my opinion, due to the presence of toxins, which, when isolated by filtration, produce an almost equal effect. Subcutaneous injection is sometimes followed by symptoms, which, however, never resemble the original disease.

Severe symptoms were produced in a healthy horse after it had received blood from a horse suffering from malaria sequelæ. This experiment is important, as it demonstrates that a more pronounced pathogenic result is obtained when the bacillus is injected into a horse which stands under the influence of the pyroplasma. The fact that the subcutaneous injection of a pure culture produces suppuration and necrosis is another indication of the pathogenicity of the bacillus in the horse. These experiments also point to the conclusion that the bacillus is not the *causa causans* of the disease described, but that the disease is due to the symbiotic effect of both, or to a secondary infection by the bacillus after the animal has been primarily affected with the pyroplasma equi.

In this symbiotic disease one part of the infection is due to a micro-organism belonging to the animal kingdom, and the other to one belonging to the vegetable kingdom. The impossibility of cultivating the former in artificial media makes it impossible to produce the disease experimentally by the inoculation of cultures.

As has been already mentioned, the pyroplasma equi has never, until now, been transmitted to healthy horses by the injection of blood, and the explanation of this failure has been already given.

When, however, the presence of the bacillus in the system of horses which show all more or less the same *post-mortem* symptoms is proved, and when this absence from horses which died from other diseases is shown, we obtain indirect evidence as to the conditions which the bacillus requires for its development. Although at one time it was thought that the presence of a specific bacterium elsewhere than in a typical disease of which it was presumably the *causa causans*, was evidence against the specificity of the bacterium, this view has not stood the test of recent experience. We know now that a micro-organism may be present in healthy animals without producing any ill effect, and that special conditions are required for its development. Such is especially the case in symbiotic diseases similar to the one under review.

One fact I have observed, that the bacillus I have described may occasionally be present in horses which die but do not show the

typical symptoms I have described, but this was rare (only two cases).

The following is a record of the bacteriological examinations of the spleens of horses dead from other diseases than the sequelæ of malaria :—

1900.				
1.	Sept. 15	.	Enteritis; <i>post-mortem</i> shortly after death.	Bacterium found not identical with the one in question.
2.	" 21	.	Pneumonia.	—
3.	" 24	.	Enteritis; <i>post-mortem</i> shortly after death.	Bacterium as in No. 1.
4.	" 26	.	Chronic enteritis; <i>post-mortem</i> ten hours later.	Nothing found.
5.	Oct. 1	.	Enteritis; <i>post-mortem</i> fourteen hours after death.	Bacterium found, but not identical with the typical one.
6.	" 3	.	Pneumonia, endocarditis, enteritis; <i>post-mortem</i> half an hour later.	—
7.	" 9	.	Colitis; <i>post-mortem</i> four hours after death.	—
8.	" 9	.	Enteritis; <i>post-mortem</i> five hours after death.	—
9.	" 10	.	Colic; <i>post-mortem</i> ten hours after death.	—
10.	" 12	.	Enteritis; <i>post-mortem</i> four hours later.	—
11. }	" 13	.	Cast horses killed; <i>post-mortem</i>	—
12. }	" 13	.	six hours after death.	—
13.	" 14	.	Experimental horse - sickness; <i>post-mortem</i> immediately after death.	—
14.	" 17	.	—	—
15.	" 18	.	—	—
16.	" 22	.	Experimental enteritis; <i>post-mortem</i> directly after death.	—
17.	" 24	.	Isolation.	Bacteria found, but not identical with the typical one.
18.	" 30	.	Experimental horse - sickness; <i>post-mortem</i> directly after death.	—
19.	" 31	.	—	None present.
20.	Nov. 1	.	Cast horse killed; <i>post-mortem</i> directly after death.	Bacteria present not identical.
21.	" 7	.	Chronic pneumonia.	—
22.	Dec. 12	.	Horse-sickness.	None present.
23.	" 17	.	Gastritis, enteritis, nephritis.	Bacteria present not identical.
24.	" 31	.	Anthrax.	Anthrax bacterium.
1901.				
25.	Jan. 5	.	Experimental horse killed with bacillus typhi abdominalis.	Bacillus typhi abdominalis.
26.	" 14	.	Experimental horse killed with bacillus coli; <i>post-mortem</i> three hours after death.	Bacillus coli
27.	" 22	.	Horse-sickness.	None present.
28.	" 24	.	—	Bacteria present not identical.
29.	" 24	.	—	Bacillus coli.
30.	Feb. 6	.	Pneumonia.	None present.
31.	" 6	.	Horse-sickness; <i>post-mortem</i> five hours after death.	Bacillus coli found.

32.	Feb. 13	.	Horse-sickness; <i>post-mortem</i> six hours after death.	Bacterium fluorescens foetidum.
33.	" 8	.	Horse killed with typhoid bacillus.	Bacillus typhi abdominalis.
34.	" 14	.	Horse - sickness; <i>post - mortem</i> twelve hours after death.	Bacteria found not identical.
35.	" 16	.	Horse - sickness; <i>post - mortem</i> twenty-four hours after death.	—
36.	" 18	.	Horse-sickness.	None found.
37.	" 19	.	—	Bacteria found not identical.
38.	Mar. 27	.	Pneumonia.	Bacillus coli
39.	" 29	.	Nephritis.	Bacteria found not identical.
40. {	" 20	:	Pneumonia and influenza.	None found.
41. {	" 20	:		

The following are the cases in which the typical bacterium was found:—

(42.) 21st September 1900. Experimental horse 25 for horse-sickness. This horse was suffering from a big abscess in the post-pharyngeal gland, which prevented the animal from eating. On *post-mortem* the spleen was found normal. Cultures revealed the presence of the typical bacillus.

(43.) 22nd September 1900. Very poor horse. *Post-mortem* showed general anæmia. Pneumonia cavernosa. Pleuritis fibrosa, spleen normal. Contains the specific bacterium.

A summary of the above observations shows that out of forty-three *post-mortem* examinations upon horses which did not show symptoms of the sequelæ of equine malaria only two cases were found to show the specific bacillus; this corresponds to 3·2 per cent. The bacillus was found in eighteen cases of typical malarial sequelæ, corresponding to 30·5 per cent., and was not found in 41 similar cases, corresponding to 69·4 per cent. of all *post-mortems* made which were bacteriologically examined. These figures do not include the *post-mortems* of horses which were killed by the injection of the typical bacillus. These statistics prove that the bacillus which I have described is not merely a casual concomitant, but is a specific factor in a certain series of pathological changes commonly found in malaria sequelæ. In cases 40 and 41 the horses were in very low condition; it may therefore be allowed that they offered a suitable medium for the development of the bacillus.

The fact that the *post-mortems* were made at varying intervals after death—even as late as twenty-four hours—proves that the invasions of the bacterium into the spleen was not a *post-mortem* phenomenon, it having been found *intra vitam*, and at all times after death in the typical cases, and not in others. Some of the bacteria found in the spleen were certainly due to *post-mortem* invasion, or rather to a preagonistic invasion. They were all closely studied, but most of them could not be identified, and as they differed one from another the study was not continued.

Agglutination.—In addition to the evidence already brought forward as to the specificity of the coccobacillus described, we have a still further proof in the fact that the serum of a horse suffering from malaria sequelæ does very often agglutinate the bacilli, when mixed with a bouillon culture. This phenomenon served in the first instance for the identification of a bacterium after it was isolated from

the spleen, and in all instances the typical precipitation of the culture took place when mixed with a corresponding serum. The serum was prepared by subcutaneous injection (horse 185) or by intrajugular injection (S.H.).

Before the serum horses were injected, their serum was tested with as many different sub-cultures from different horses, and then it was found that the normal serum did not agglutinate the cultures. One single injection of a culture is sufficient to produce the agglutinating power. Further, the injection of the filtrate of a bouillon culture produces an agglutinating serum.

In highly immunised horses this power is so high that agglutination takes place very rapidly after the mixture with the culture. The agglutination was tested in the proportion of one part of the serum to one hundred parts of bouillon culture; for this purpose, to 5 cc. of bouillon culture one drop of serum was added. But the phenomenon could still be observed when the dilution was made 1:1000; the precipitation took place after some time. In the proportion 1:100, the phenomenon began shortly after the mixture, and was generally complete in one hour; it rarely took two hours. The bacteria collected in big flakes and subsided, leaving the bouillon as clear as sterile bouillon. The clumps massed closely together, and it was only by shaking the test tube vigorously that a mass could be whirled up. In order to ascertain whether apparently healthy normal horses possessed an agglutinating serum, thirty-four horses were tested at three different times; when it was found that only two horses possessed a serum which agglutinated in a strength of 1:100. I also tested the specific serum on other cultures than the one in question, namely, on chicken cholera, on *bacillus coli communis*, *bacillus typhi abdominalis*, and on all the different bacteria isolated from the spleens of the different horses which on microscopical examination and culture proved not to be identical with the one in question. In no instance did the serum in a strength of 1:100 show an agglutinative power. Hence the conclusion that the serum obtained by the injection of the bacterium of malaria sequelæ is specific for this one bacterium.

In order to ascertain the time required by a horse to produce an agglutinating serum, a horse (No. 90) was injected with 5 cc. of a bouillon culture into the jugular. This horse was tapped before the injection, and on each day after the inoculation up to the twelfth day, and the different samples of sera were then tested. It was found that from the fifth day the precipitation began, that on the sixth, seventh, and eighth day it continued to increase in intensity, but after the eighth day there was no further increase. The horse appeared to retain the agglutinating power for a considerable time. One horse (S.H.) was repeatedly tested for a period of a year, and its serum was always found active. Serum kept in test tubes at ordinary temperature, however, had lost this power after four months.

The phenomenon of agglutination is a sign of infection by the bacterium which produces it, and can therefore be utilised for diagnostic purposes. It was therefore utilised in the case of several horses which were suspected to be suffering from malaria sequelæ, or which were actually suffering from bilious fever.

(1.) Horse 34 (14 of the S.U. list). This horse was found suffering

from malaria on 2nd March. On 10th March blood was collected in test tubes and allowed to coagulate. The next day the serum was tested on all the previously isolated cultures of the bacillus in question; it was found that all of them were typically agglutinated. The horse died on the night 11th March, when the bacterium was isolated from the spleen.

(2.) Horse 1050 (No. 11). This horse died on the night 3rd January 1901. During the 3rd January blood was collected. On 7th January the serum was tested on a bouillon culture, and it was found that it agglutinated very rapidly and typically. The bacillus was obtained from the spleen.

(3.) Horse. Dr T. English mare. On the 5th April this horse showed symptoms of malaria and the pyroplasma equi was found. The horse was treated, and recovered. But as, from previous experience, symbiotic infection with the bacillus of malaria sequelæ might be anticipated, the horse was tapped daily, and the serum tested for agglutination. The serum of the first day and the following day showed no agglutinative power whatever. The pyroplasma had disappeared. On the 16th April the serum of the horse showed distinct agglutinative power, and the power increased during the following days.

(4.) Horse S.A.C. 585. (Case 17). The serum of this horse was taken two days previous to its death on the 13th March 1902, and was tested on the 15th March. The precipitation was distinct. The bacillus was isolated from the spleen.

(5.) 25th March 1902. A horse of the S.A.C. belonging to Colonel St., showed distinct symptoms of biliary fever. It was treated by Captain Christy, and recovered. Captain Christy was good enough to put some serum at my disposal on the seventh day after the animal had entered the sick lines. This serum did not agglutinate. The animal was tapped again on the 29th March, and tested, when it was found that it agglutinated promptly.

(6.) 15th April 1902. Horse 24 of the S.A.C. suffering from bilious fever, but recovered. The case was diagnosed by Captain Christy and tested. After recovery the serum was tested, and the test repeated with serum taken on the 17th April. No reaction whatever took place.

(7.) 3rd June 1902. Horse belonging to the T.C.S. Company. This animal showed all the symptoms of biliary fever. The pyroplasma equi, however, was not found. Blood was collected, but the serum did not agglutinate. On the 13th June blood was obtained, the animal having recovered, and the serum was found to agglutinate promptly.

This is all the evidence which I have been able to collect, but from this evidence we may conclude that an animal which is suffering from malaria sequelæ produces an agglutinative serum. For production of this serum a time must elapse of at least five days between the date of infection and the appearance of the agglutinins in the blood. From Cases 3 and 7 we may gather that even horses which suffer from malaria fever and which recover may become infected by the bacterium of the sequelæ, and that they do not necessarily succumb. This is further evidence of the symbiotic, or rather, secondary infection in bilious fever.

Case 6 proves that there may be infection with the pyroplasma equi, but no secondary infection.

Cases 3 and 7 illustrate also the observation quoted above, where, out of thirty-six apparently healthy animals, three had a distinct agglutinating serum. These two animals probably had some time in their earlier life suffered from bilious fever and the sequelæ, and recovered, and the sera had retained the agglutinating power.

We may further conclude that symbiotic and secondary infection due to the pyroplasma equi on the one side, and to the bacterium on the other side, are not necessarily fatal.

To what recovery is due, I am unable to say. The serum of a horse which has received several injections of bouillon cultures of the bacillus of malarial sequelæ, and which is highly agglutinative, has no noticeable bactericidal action *in vitro*. Agglutinated cultures when transplanted into fresh agar developed even after a contact of fourteen days. I am not yet in a position to state what effect the serum of a highly immunised animal has on infected animals, when used as a curative agent.

It remains now to trace the origin of the bacterium, and how it comes into the animal's body. The idea that the infection takes place from the intestines will at once suggest itself. Seeing that it is, so to say, constantly found in the biliary fever cases, one is entitled to believe that the bacillus may be fairly constantly present in the horse, but that it enters the animal's blood and becomes pathogenic when the vitality of the antitoxic and bactericidal power of the animal juices is reduced. I was unable to isolate the bacillus by plate cultures on gelatine or agar from the dung, even though I experimented both with healthy horses and with horses whose sera agglutinates cultures of the bacillus.

The most common bacterium was a bacillus coli communis which corresponds to the typical bacillus coli communis of the text books.

Summary of Conclusions.

- (1). Equine malaria or biliary fever is due in the first instance to an invasion by the pyroplasma equi.
- (2). The pyroplasma equi predisposes to a symbiotic and secondary infection due to a specific bacillus.
- (3). The secondary infection can be traced by the agglutination test *intra vitam*, provided that the infection has existed for five days.
- (4). The secondary infection can take place even after complete recovery from the biliary fever.
- (5). The secondary infection is not invariably fatal.
- (6). When a healthy horse possesses agglutinating serum it is probable that it has suffered at some time from equine malaria.

SOUTH AFRICAN HORSE-SICKNESS.

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THE worst part of the horse-sickness season of 1903 being over, I propose to give an account of *post-mortems* made on forty-eight cases of this disease, these cases all occurring in the space of about two

months. Attached will be found a table in which an endeavour has been made to classify the cases according to the most prominent lesions found on *post-mortem* examination. The classification is only a rough one, as one cannot draw a fine distinction.

In the attached table it will be seen that Classes I., II., and V. differ only in the extent of the bowel lesions. In Class I. the only marked bowel lesion is a very intense inflammation of the mucous membrane of the pyloric portion of the stomach. In Class II. the inflammation of the mucous membrane is present in the small and large intestine, also to a varying extent. In these cases the inflammation of the mucous membrane is in the majority of cases most intense at the pelvic flexure of the large colon and also in the cæcum. At these parts the inflamed membrane is frequently of a blood red or purple colour, in many cases as intense as that of the pyloric membrane of the stomach. A quantity of gravel is frequently present in the cæcum and large colon, but this is a common occurrence in cases other than horse-sickness, and has no direct bearing on the disease. Occasionally, but rarely, there are noticeable in the mucous membrane of the intestines small, slightly depressed, ulcer-like lesions which are small areas of surface necrosis.

Before proceeding any further I should like to make a particular point of the fact that not in one single case of the forty-eight *post-mortemed* was an intense inflammation of the mucous membrane of the pyloric portion of the stomach absent. This organ was in every case made the object of very careful inspection, in view of the fact that this particular lesion was said by some observers not to be a common one in South Africa. I was fortunate enough to be present at *post-mortems* made on several cases of experimental horse-sickness at the Royal Veterinary College, London, by Prof. M'Fadyean, and the invariable presence of this lesion was very striking.

A close examination of the pyloric mucous membrane of the stomach in these cases *post-mortemed* by myself was not necessary to detect this lesion, although occasionally the membrane was covered by a thick layer of a white mucus-like material, which, however, is easily removed by gently passing the edge of the knife over the part, disclosing the intensely inflamed membrane beneath. The colour of this inflamed pyloric membrane varies from blood to purple, and always suggests that some very irritant poison has been at work. The inflammation of the mucous membrane of the small intestine usually takes the form of a slight muco-enteritis, but occasionally it is very much more marked, and in the form of ring-like bands (zebra marking). In some cases blood extravasations are present. I have never met with a case of very intense inflammation of the mucous membrane of the floating colon, and when present at all the colour of the membrane is of a rose pink.

In Classes III., IV., and VI., the intestinal lesions in the majority of cases were more marked than in Classes I., II., and V., and the lesions in the thoracic cavity correspondingly less marked.

Symptoms in Classes I., II., and V.—In these cases the animals were usually not brought to the sick lines until they had commenced to show symptoms of distress, *viz.*, accelerated or difficult breathing; and death usually took place within a few hours, the disease being so far advanced as to render treatment hopeless. Other symptoms shown are: intense

Class I.	Class II.	Class III.	Class IV.	Class V.	Class VI.
<p>1. Pneumonic symptoms and rapid death after their onset.</p> <p>2. Discharge of frothy serum after death from nostrils. (Sometimes also before death.)</p> <p>3. Lungs extremely oedematous. A quantity of clear amber-coloured gelatinous exudate on their surface.</p> <p>4. A clear amber-coloured or blood-tinged exudate in pleural cavity.</p> <p>5. A clear amber-coloured or blood-tinged exudate in pericardial cavity.</p> <p>6. A very intense inflammation of the mucous membrane of the pyloric half of the stomach.</p>	<p><i>Ditto I.</i></p> <p>Inflammation of the mucous membrane throughout the intestinal tract.</p>	<p>1. Death not usually so rapid after onset of clinical symptoms.</p> <p>2. Oedema of the tissues of the neck, and filled condition of the supra-orbital cavities usually present.</p> <p>3. Slight oedema of lungs, and small quantity of gelatinous exudate or absence of oedema.</p> <p>4. Pleural exudate as in Class I.</p> <p>5. Pericardial exudate as in Class I.</p> <p>6. Very intense inflammation of the mucous membrane of the pyloric half of the stomach and throughout the intestines.</p>	<p><i>Ditto III.</i></p> <p>Inflammation of the mucous membrane of the intestines.</p>	<p><i>Ditto I.</i></p> <p>Intense inflammation of the mucous membrane of the large colon only.</p>	<p><i>Ditto III.</i></p> <p>Intense inflammation of the mucous membrane of the pyloric half of stomach and large intestine only.</p>
<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Roan Mare 6 10/9/03 11/9/03</p> <p>2. Bay Geld. 6 14/2/03 14/2/03</p> <p>3. Bay Geld. 6 16/2/03 17/2/03</p> <p>4. Ch. Mule 19/2/03 19/2/03</p> <p>5. — 22/9/03 22/9/03</p> <p>6. Bay Geld. 7 22/9/03 22/9/03</p> <p>7. — 15/9/03 17/9/03</p>	<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Bay Geld. 7 21/9/03 21/9/03</p> <p>2. Br. Geld. 7 22/9/03 22/9/03</p> <p>3. Bay Mule 22/9/03 22/9/03</p> <p>4. — 22/9/03 22/9/03</p> <p>5. — 26/2/03 26/2/03</p> <p>6. Bay Geld. 7 26/2/03 26/2/03</p> <p>7. Bay Mare 6 26/2/03 26/2/03</p> <p>8. Ch. Mare 7 26/2/03 26/2/03</p> <p>9. Bay Geld. 7 11/9/03 12/9/03</p> <p>10. Ch. Geld. 7 11/9/03 12/9/03</p> <p>11. — 16/9/03 16/9/03</p> <p>12. Bay Geld. 7 16/9/03 16/9/03</p> <p>13. Roan Geld. 7 19/9/03 19/9/03</p> <p>14. Ch. Geld. 7 7/4/03 7/4/03</p>	<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Br. Mare Agel 19/9/03 21/9/03</p> <p>2. Bay Geld. 6 22/2/03 24/2/03</p> <p>3. Br. Mare 6 26/2/03 26/2/03</p> <p>4. Grey Mare 6 27/2/03 27/2/03</p> <p>5. Br. Mare 6 27/2/03 27/2/03</p> <p>6. Ch. Geld. 6 27/2/03 27/2/03</p> <p>7. Br. Mare 6 27/2/03 27/2/03</p> <p>8. Grey Geld. Agel 17/9/03 17/9/03</p> <p>9. Bay Geld. 7 17/9/03 17/9/03</p> <p>10. Bay Geld. 6 18/9/03 18/9/03</p>	<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Roan Mare 7 22/9/03 22/9/03</p> <p>2. Ch. Mare 7 22/9/03 22/9/03</p> <p>3. Br. Mare 7 22/9/03 22/9/03</p> <p>4. Bay Geld. 7 22/9/03 22/9/03</p> <p>5. Br. Mare 6 22/9/03 22/9/03</p> <p>6. Bay Geld. 6 22/9/03 22/9/03</p> <p>7. Bay Geld. 6 22/9/03 22/9/03</p> <p>8. Ch. Geld. 7 22/9/03 22/9/03</p> <p>9. Bay Geld. 7 22/9/03 22/9/03</p> <p>10. Grey Geld. Agel 21/9/03 21/9/03</p>	<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Bay Geld. 7 22/9/03 22/9/03</p> <p>2. Br. Mare 6 22/9/03 22/9/03</p> <p>3. Bay Geld. 6 22/9/03 22/9/03</p> <p>4. Bay Geld. 6 22/9/03 22/9/03</p> <p>5. Br. Mare 6 22/9/03 22/9/03</p> <p>6. Bay Geld. 6 22/9/03 22/9/03</p> <p>7. Bay Geld. 6 22/9/03 22/9/03</p> <p>8. Ch. Geld. 7 22/9/03 22/9/03</p> <p>9. Bay Geld. 7 22/9/03 22/9/03</p> <p>10. Grey Geld. Agel 21/9/03 21/9/03</p>	<p>Colour, Sex, Age, Admitted, Died.</p> <p>1. Bay Geld. 7 22/9/03 22/9/03</p> <p>2. Br. Mare 6 22/9/03 22/9/03</p> <p>3. Bay Geld. 6 22/9/03 22/9/03</p> <p>4. Bay Geld. 6 22/9/03 22/9/03</p> <p>5. Br. Mare 6 22/9/03 22/9/03</p> <p>6. Bay Geld. 6 22/9/03 22/9/03</p> <p>7. Bay Geld. 6 22/9/03 22/9/03</p> <p>8. Ch. Geld. 7 22/9/03 22/9/03</p> <p>9. Bay Geld. 7 22/9/03 22/9/03</p> <p>10. Grey Geld. Agel 21/9/03 21/9/03</p>

congestion of the conjunctival membranes, and the invariable presence on them of red or purple petechial spots. This condition of the membranes is a characteristic feature. The pulse is always quickened, very weak, or almost imperceptible. The temperature is usually high (105°), but in cases where death is imminent it may be apparently normal, though in reality running down just before death.

Frequently about half-an-hour before death the animal commences to discharge from both nostrils huge quantities of a yellowish or white frothy serum, but if this does not occur before death it invariably does after death in this class of case. Usually these are the only noticeable symptoms.

On *post-mortem* one finds the lungs to be greatly enlarged from œdema, the serous fluid being present in such quantities as to run away on section. This fluid is turned into froth in the lungs themselves, as well as on coming in contact with the atmospheric air, this being due to the churning process of respiration. A quantity of a clear amber-coloured gelatinous exudate is almost invariably present on the surface of and between the lungs, also intersecting the lobules of the lungs near the surface. A dropsical transudate is almost invariably present in the pleural and pericardial cavities. The quantity varies from a few ounces up to 2 or 3 pints, and is usually of a clear amber colour, free from any signs of putridity; occasionally it is blood tinged. The intestinal lesions vary in extent, as shown above.

Symptoms in Classes III., IV., and VI.—Frequently in a careless examination the only symptoms that would be observable would be dulness and a tendency to lie down. This tendency to lie down is always present, and indicates a condition of uneasiness akin to colic. If a closer examination of the patient be made the conjunctival membranes will be found to be very dark and intensely injected, showing petechial spots. The pulse will be found to be quickened and very weak. This weakness of the pulse I put down in the majority of instances to the mechanical interference of the dropsical transudate with the heart's action, this being frequently present in such quantity as to be retained under pressure within the pericardial membrane. The temperature is always raised unless death be imminent. Death usually does not take place so rapidly as in the pneumonic forms. The patient may live for several days, or death may take place within a few hours of clinical symptoms being noticed by the ordinary layman. Probably, however, if observation had been carried out for several days previously to the animal being brought to the sick lines by an experienced eye, the animal would have been noticed not to be in his usual health.

The symptoms shown in this class of case are always indicative of intestinal trouble, the animal sometimes grunting and turning his head towards his abdomen. In fact, the patient shows the symptoms of enteritis.

It is not always easy to diagnose cases of this class in their early stages, but the condition of the membranes is a good guide. In this country cases of fever with discoloured membrane are common, being probably malarial fevers, but it is possible to draw the conclusion that more animals than is at present supposed become affected with horse-sickness, but recover in the early stages of the disease.

Other symptoms more frequently shown in this class of case than in the pneumonic form are œdema of the tissues of the neck and filling of the supraorbital cavities, so that they become convex instead of concave. When this œdema of the tissues of the neck is present in a minor degree, or so as not to be observable without manipulation, it can be frequently noticed by a peculiar, slightly indurated, cord-like feeling of the sternomaxillaris muscle; and later the tissues of the neck, instead of having the loose feeling when the front of the normal neck is grasped and moved from side to side, seem closer knitted together and firmer, and the trachea particularly prominent.

At the *post-mortem*, as previously mentioned, the lesions in the abdomen are usually more marked than the corresponding lesions in the acute pneumonic form. There is usually present slight œdema of the lungs and tissues on the chest wall. The lesions of the pleural and pericardial cavities are the same as in cases of the pneumonic forms, and I have never yet met with a case where there was no fluid in either of these cavities.

These two forms of horse sickness, viz., that in which the marked symptoms are pneumonic, and that in which the marked symptoms are abdominal, appear to me to be distinct enough to warrant one speaking of a pneumonic and an intestinal form. I have had no experience with the dikkop form, which I presume is quite distinct from the form in which one gets œdema of the tissues of the neck, but no swelling of the head except the filling of the supraorbital cavities. I should have mentioned that in the intestinal form, or, better, gastro-enteric form, the animal usually lives long enough to lose condition, whereas in the other form animals at the time of death are usually in excellent condition.

The liver in neither form appears to show characteristic lesions, nor do the kidneys. In only one case did I find the spleen distinctly enlarged, in which case it was quite twice its normal size. In other cases it may have been slightly enlarged and heavier, but, not having had scales to hand, it is useless to assert that the spleen is usually enlarged.

Treatment.—I must admit that I have not been very successful in the treatment of the disease. In the majority of cases when the patient is first seen the disease is too far advanced to allow any hope of successful treatment. The gastro-enteric form, on account of its slower progress, is more amenable to treatment. I have used anti-septics given as drenches, and also injected subcutaneously and intratracheally. Carbolic acid as an emulsion with linseed oil may be given in $\frac{1}{2}$ ii doses twice or thrice daily, but drugs given by the mouth do not give satisfactory results. Carbolic acid may also be injected into the trachea with glycerine as the vehicle. This causes a discharge from the nostrils from irritation of the tracheal mucous membrane, but causes no distressing symptoms from its irritation, and the discharge soon ceases after the injections are discontinued. Intratracheal injections of hydrarg. biniod. and pot. iod. have afforded me the most satisfactory results. The injection of this mixture also causes discharge from the nostrils, but the cause of the discharge is purely local. The percentage of recoveries by any method of treatment is small, provided that there is no doubt as to the case being one of horse sickness. Out of forty-eight cases, four recovered under

treatment, and one was well on the road towards recovery, but had a relapse and died. The following is a brief account of these cases:—

I. Bay gelding, six years old, admitted 12th March 1903, showing the following symptoms: conjunctival membranes extremely injected, showing purple petechial spots; great dulness and difficult breathing; supraorbital cavities filled; swelling of neck, due to œdema of the tissues. Temperature, A.M., 104° ; P.M., 104.3° ; pulse, 65; respiration, 24.

Treatment.—One injection intratracheally morning and evening of acid. carbol. pur. zj , glycerine zjs . This caused a clear discharge from the nostrils, but no coughing or injection.

13th March 1903.—Membranes not so deep a colour; petechiæ still present. Temperature, A.M., 103.6° ; P.M., 103° ; pulse, 60; respiration, 20. Having run out of carbolic acid, two injections of the following administered intratracheally: hydrarg. biniod. grs. iii, pot. iod. grs. xv, aquæ zjs . Discharge from nostrils continues.

14th March 1903.—Temperature, A.M., 102.2° ; P.M., 102° ; pulse, 52; respiration, 20. Condition about the same. Treatment continued, but dose of hydrarg. biniod. reduced to gr. i at each injection.

15th March 1903.—Temperature, A.M., 101.6° ; P.M., 100.2° ; pulse, 40; respiration, 18. Decided all round improvement; petechial spots disappeared. Œdema of neck dispersing. Intratracheal injections continued.

16th March 1903.—Temperature, A.M., 100.6° ; P.M., 100° ; pulse, 40; respiration, 18. Animal brighter; feeding moderately. Injections discontinued. From this date improvement continued. Belladonna administered to stop the nasal discharge. The patient was discharged cured on the 1st April 1903.

II. Bay gelding, aged, admitted 18th March, 1903.—This case was practically the same in its course as case I., and also treatment, except that hydrarg. biniod. and pot. iod. were used from the commencement. The patient was discharged cured on the 6th April 1903.

III. Bay mare, seven years old, admitted 18th March 1903.—Symptoms and treatment precisely similar to the previous cases. Continued to progress satisfactorily up to the 26th March 1903, when she was suddenly taken worse, and died on the 28th March 1903.

IV. Chestnut gelding, seven years old, admitted 17th March 1903.—Discharged cured on 13th April 1903. Symptoms, treatment, and course of disease similar to previous cases. This animal had a large hard swelling at the seat of injection in the neck, due to some of the liquid injected having got into the space between the skin and trachea. This swelling, however, gradually disappeared without any unfavourable symptoms.

V. Mule, admitted 20th March 1903, showing following symptoms: Temperature, A.M., 107° ; P.M., 105.6° ; pulse almost imperceptible; heaving respiration accompanied by grunting. Œdema of tissues of neck. Supra-orbital cavities filled. Two intratracheal injections administered, each containing hydrarg. biniod. grs. iii, pot. iod. grs. xv, aquæ zjs . The injection caused the mule to cough; at the same time brought up about one pint of white frothy material.

21st March 1903.—Temperature, A.M., 104.6° ; P.M., 105° ; still

grunting, and breathing heavily. Treatment continued. Case given up as hopeless. Besides the intratracheal injections, the same dose was injected at the same time as the second intratracheal injection on either side of the neck subcutaneously, so that on this day the patient received altogether hydrarg. biniod. grs. xii.

22nd March 1903.—Temperature, A.M., 104°4'; P.M., 103°6'; heaving respiration; pulse very weak. Two injections intratracheally.

23rd March 1903.—Temperature, A.M., 103°; P.M., 102°. Two injections.

24th March 1903.—Temperature, A.M., 101°6'; P.M., 100°4'; respiration, 32; pulse very weak. Lost a lot of condition.

25th March 1903.—Temperature, A.M., 100°2'; P.M., 99°. From this date the mule continued to improve, and is now practically fit and rapidly putting on condition. The only reason that the patient is still in the sick lines is that the injection on one side of the neck caused a large area of skin to slough, and that is not yet quite healed.

This was a remarkable case, and I feel convinced that recovery was due to the almost poisonous doses of hydrarg. biniod. administered. Another peculiar feature is the fact that whereas the hydrarg. biniod. injected into the trachea caused no serious damage except a discharge from the nostrils, when injected under the skin it caused the latter to slough. It only proves how very tough and resisting the tracheal mucous membrane must be.

Cause and Prevention.—Several theories have been put forward as to the cause of this disease, but up to the present nothing is definitely known as to the actual causal organism, if organism there is; and there seems to be little doubt that the disease is caused by a micro-organism, but all the efforts with the present powers of the microscope have been unavailing to discover it. Animals may be readily infected by inoculation with blood of a horse affected with the disease, the period of incubation in these experimental cases being almost invariably eight or nine days. Probably the period between infection and death is the same in natural cases, since the *post-mortem* lesions in experimental and natural cases are similar. It is said that if animals are prevented from grazing after sundown, and in the morning before the sun is well up and has dried the veldt, the disease will be prevented, and also if a nosebag be worn at night for a similar purpose. But in my opinion this theory is absolutely untenable, and is not borne out by facts. The animals under my charge after the first few cases had occurred were prevented from grazing at all, and were not even allowed to water until within the suggested preventative hours, yet the disease was in no way checked. I have also heard it expressed by other observers who have experienced several seasons that, even when these commonly suggested precautions were taken, cases occurred just as frequently. There is no doubt that some low-lying parts are very infective, especially where water and herbage is abundant.

The method of infection also is not known, but the mosquito inoculation theory seems to fit in best with observed facts in connection with the disease. Animals kept in stables and not taken out at night contract the disease far less commonly. The idea amongst farmers seems to be that the disease is contracted one night,

and the animal dies the following day, and little attention seems to be paid to where the animal happened to be, and what he was doing a reasonable time beforehand. The inoculation theory fits in with the observed fact that the disease is more commonly contracted in marshy districts and also at night; and the fact of the horse sickness season being practically over with the first severe frost would be difficult to explain, except for the possibility of the causal organism resting in some secondary host during the winter months in a state of inactivity.

DERMOID CYSTS.¹

By J. R. U. DEWAR, F.R.C.V.S., Royal (Dick) Veterinary College, Edinburgh.

THE subject of dermoid cysts, whether dentigerous or not, has been very little studied by English veterinary surgeons. In fact, the literature of the subject is of the most scanty description. In looking up the subject, I have searched the *Veterinarian* back to the thirties, the *Veterinary Journal* since its commencement, and some volumes of the *Veterinary Review* without finding much of importance. Within the past few years rather more attention has been directed to them, but it seems certain that numbers of cases that must be observed are allowed to slip away into the past without being recorded, consequently without inciting to further study.

Etiology.—The etiology or causation of these cysts or congenital abnormalities has been very much disputed, and it is doubtful whether their "raison d'être" has yet been satisfactorily explained.

Dr Payne² rejects the hypothesis of the older writers, that they are the remains of blighted embryos included during development in the more perfectly normal individual.

Without being at all dogmatic, he states that "it would rather appear as if a portion of embryonic tissue from the upper and middle germinal layers became displaced at an early period of development."

Somewhat similar views were very generally held. Thus we find Professor Mettam, in describing two dentigerous cysts,³ stating: "It is generally considered that this form of cyst is due to an error in development; that in the closure of the branchial clefts—in this case of the first—a small portion has been separated off, and along with it part of the tooth-bearing segment of the palato-pterygoid division of the arch bounding the cleft has been included." He, however, admits that this theory does not account for the presence of cysts containing teeth in the ovary, testicles, and distant parts of the body. These, he states, "are generally considered as forms of teratomata, or embryonic tumours." But we fail to perceive why a cyst containing teeth and other tissues should be held to have an entirely different origin if situated in the temporal region, from what it would have if in an ovary.

¹ A Paper read before the South Durham and North Yorkshire Veterinary Medical Association.

² "Quains Dictionary of Medicine," 1883.

³ "Veterinarian" for May 1899, p. 309.

Möller¹ ascribes these dentigerous cysts in the cephalic region to similar causes: "to the development of embryonic branchial arches and clefts."

Now Professor Max Wilms² seeks to prove that these hypotheses are all wrong. He states that the dermoid cysts found in the ovaries, testicles, etc., are rudimentary embryos, and that they are probably developed from fertilised polar bodies.

Dr Beard, in a short review of Professor Wilms' work,³ points out that "primary germ cells . . . are now for the first time recorded as present in situations other than in the sexual glands," and again, "the existence of vagrant primary germ cells is established," and "my position regarding the embryomas is this, that they may occur in almost any part of the body, but that naturally they are far more numerous in ovary and testes. They always arise from persistent primary germ cells."

Dr Beard seems to hold that the formation of ova from epithelial tissue is a myth, and that there is no such thing as germinal epithelium covering the ovary. The theory is that after the fertilisation of the ovum, after the union of one sperm cell and one germ cell, the normal process of development of the segmentation nucleus leads to the formation of a definite number of primary germ cells, of which one goes to form an embryo, while the rest migrate into it to furnish its sexual products. And that it is owing to the failure of these germ cells to all collect into the ovary or testicle, as the case may be, and to the presence of vagrant or wandering cells amongst the somatic cells or cells of the body proper, that the formation of these dermoid cysts or embryomas is due.

As far as I can make out, the contention is that unfertilised germ cells are in some of the lower forms of vertebrate life found in regions of the body apart from the sexual glands, and that under exceptional circumstances—as yet unexplained—they may, and do, undergo a certain amount of development. That the probability is that the same thing occurs in the higher forms of vertebrate life, and that bone, nerve matter, teeth, and rudiments of fore limbs are sometimes formed, while hair, sebaceous and sweat glands seem always present; in fact, that these embryomas represent more or less of the anterior end of an embryo.

It is recognised by embryologists and biologists that in some of the lower forms of animal life a certain amount of development is possible in what might be termed maiden ova, that is to say, the development of germ cells may start and go on to the extent of forming the more complex animal tissues without fertilisation, without impregnation having taken place,—parthenogenesis it is termed,—although the process becomes arrested far short of the formation of a complete normal individual.

Assuming for the time that this hypothesis is correct, it would certainly explain far more satisfactorily than any hitherto promulgated the occurrence of these tumours in different parts of the body, and of tissues in regions far distant from their normal situation.

¹ Möller's "Operative Veterinary Surgery." Dollar's Translation, p. 96.

² Max Wilms. "Die Krankheiten der Eierstöcke" (by Martin), p. 576.

³ Dr Beard. "Anat. Anz.," Vol. XVIII., p. 478.

But it will undoubtedly be required to stand the test of hostile criticism. There is no doubt that many of these abnormal developments met with in the temporal region of horses are true cysts, and not merely odontomas, or dental tumours, yet it is doubtful if they always contain hair and sebaceous and sweat glands. At least it can be said that in some of them the hair requires a good deal of demonstration, and is not readily evident to the naked eye.

In our animals the dermoid cysts of the testes are much more familiar than those of the ovaries, probably because the castration of the male is common, permitting of the examination of these organs, while ovariectomy is comparatively rare, and in those testicular dermoids hair is generally very conspicuous. Two of these are very well described in the *Veterinary Journal* for July 1901, p. 22, by Mr H. Taylor, and every veterinary surgeon who has had much experience in castration must have met with them, yet it is surprising how little attention they have received in English veterinary literature. The first reference to them I have been able to find is in the *Veterinarian*, Vol. XXXIII., p. 358. It is a short summary of a paper by M. J. Macorps, translated from the *Annales de Médecine Vétérinaire*, Brussels. The author states that he has met with fourteen cases of temporal dentigerous cysts, one in a foal ten months old, nine in horses from three to five years old, and four in aged horses. He states that the operative removal of a single tooth in each case cured thirteen of them in about ten days. The fourteenth had to be operated on twice in three months, and at each operation a tooth was extracted. The crowns of these teeth all pointed upwards, the fangs downwards. In the *Journal of Comparative Pathology and Therapeutics*, Vol II., p. 152, an interesting case is recorded by the late Professor Walley.

The *Veterinarian*, Vol. LXIX., p. 411, contains a translation from the *Revue Vétérinaire*, of a case related by M. Gauthier of a cyst the size of a hen's egg at the base of the left ear of a horse four and a half years old. It contained a tooth of the size and appearance of a newly erupted molar, attached to the squamous temporal, from which it was removed by a gouge and mallet.

The *Veterinarian*, Vol LXXII., p. 309, contains two cases narrated by Professor Mettam. In the *Journal of Comparative Pathology and Therapeutics*, Vol. XIII., p. 174, Professor Hobday describes a case in a well-bred, four-year-old filly, one tooth resembling a molar being removed.

Cadiot and Dollar—*Clinical Veterinary Medicine and Surgery*, p. 1. Here a pretty full description of them is attempted, the most lengthy I have found in English veterinary literature.

Reference is made to an attempt at classification of the cysts according to the position of the contained teeth, but in my case No. 1, while one tooth had its crown towards the centre of the mass, another had the crown directed away from it.

While Cadiot recommends operative interference as usually advisable, and the operation as generally simple, the removal of the tooth—usually a single tooth—easy, and recovery rapid, the possibility of risk is pointed out should a tooth be firmly fixed in the bone, and reference is made to a case of Degive's in which the tooth extended through the walls of the cranium.

The cyst which is the immediate cause of this paper being written was situated on the right side of the head, immediately over the auditory process of the petrous temporal bone, and in a straight line between that process and the crest of the occiput. It is therefore placed in the triangular space formed by the ridge which connects the zygomatic process of the squamous temporal with the occipital crest in front, the auditory process of the petrous temporal below, and the ridge which runs from the crest of the occiput to the styloid process behind. This space has undergone considerable expansion. On the left side the measurement from the most posterior point of the petrous temporal, straight across the auditory process to the zygomatic ridge, is 4.8 cm. (about 1 $\frac{1}{2}$ inches), while on the right side it is 6.4 cm. (about 2 $\frac{1}{2}$ inches). After boiling and cleaning, so that nothing is left but

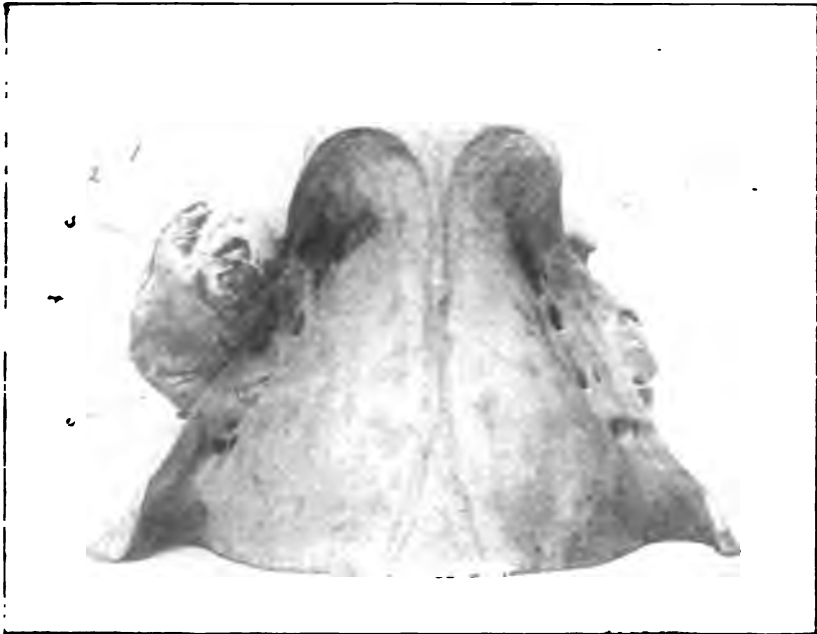


FIG. 1.

1. Rounded summit of larger tooth. 2. Fang of smaller tooth. 3. Dark roughened patch, probably due to operation. 4. Thin lamella of bone. 5. Deeper seated dentine showing through.

hard tissues, the cyst is seen to be well circumscribed. It measures 5.7 cm. (about 2 $\frac{1}{4}$ inches) in a line downwards from the occipital crest, 4.4 cm. (about 1 $\frac{3}{4}$ inches) from before backwards, and only 3.4 cm. (about 1 $\frac{1}{4}$ inches) from right to left at its upper part; although, lower down, opposite its projection into the cranial cavity, it measures in the same direction 6 cm. (about 2 $\frac{3}{8}$ inches), its greatest diameter.

The most prominent part of the cyst projects laterally 4.5 cm. (about 1 $\frac{3}{4}$ inches) outwards from a point 1.3 cm. (about $\frac{1}{2}$ an inch) below the junction of the zygomatic ridge and the ridge which connects the occipital crest and the styloid process. It is covered by a thin lamina

For the illustrations I am indebted to my colleague Prof. Dunstan.

of bone, as if due to the expansion of part of the pyramidal process of the squamous temporal, and here and there, even where not removed in the cleaning, this has all been absorbed, so that the dentine within is showing through. In front of the zygomatic ridge there are two perforations, the largest of which measures almost 1 cm. (about $\frac{3}{8}$ of an inch) in its long diameter.

Inside the cranium the cyst has led to considerable deformity, the centre of which is on the line or sharp ridge of bone between the positions of the cerebrum and cerebellum, to which the tentorium cerebelli is attached. From a line drawn straight through the centre of the cranial cavity, the nearest part of the cyst is only 1.8 cm. (about $\frac{1}{4}$ of an inch) distant, while at the same point on the opposite side the wall of the cranium is 2.8 cm. (about $1\frac{1}{8}$ inches) distant.

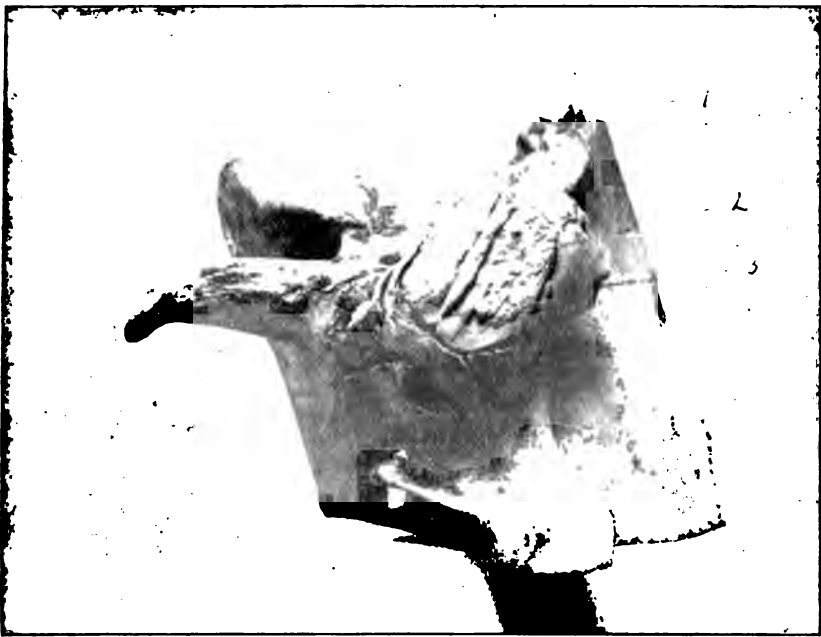


FIG. 1A. (Same Case as Fig. 1.)

1. Crest of occiput. 2. Outer surface of smaller tooth, fluted, crown lowermost—one from which the thin fenestrated lamella of bone has crumbled away. 3. Thin lamella of bone.
4. External auditory meatus.

More of the cyst projects into the cerebellar cavity than into the cerebral, what is evidently the crowns of two teeth being exposed without any covering of bone over them. The distortion is not so great on the cerebral side of the tentorial ridge, but in it there are two perforations 1.3 cm. (about half an inch) apart, through which the dentine is exposed.

Externally the auditory process is displaced downwards, flattened from above, and expanded from side to side. Its upper margin, which is normally incomplete in the foal, has never been united, and instead the edges of the process of bone have been forced some distance apart. The auditory canal is quite pervious. On examining the cyst or

dentigerous tumour itself after it had been cleared of all soft tissues, it was found that the thin lamina of bone covering it was already partly removed, and was so thin and friable that little more was required to permit of a tooth being pulled out from its outer aspect. It is a comparatively small tooth, measuring 4.2 cm., by 3 cm., by 1.6 cm. (about $1\frac{5}{8}$ by $1\frac{3}{16}$ by $\frac{5}{8}$ inches), and weighs about 12 grammes (3 drams 8 grains). It is undoubtedly a molar, or rather like a half molar, is fluted or grooved, and has enfoldings of the dental substance. The root or fang has so many enfoldings that it looks like several superimposed lamina, and is as easily made out—as is also the crown—as in a normal tooth. Its position was nearly vertical in the cyst, the crown being downwards,—deepest. Situated to the inner side of the tooth just described, but separated from it by an incomplete lamella of bone, was a second tooth. It is somewhat ovoid in shape, flattened slightly



FIG. 1B. (Same Case as Figs. 1 and 1A.)

1. Left styloid process, tip broken off. 2. Tentorium cerebelli displaced a little to the left.
3. Crown of tooth exposed projecting into the cerebellar cavity. 4. Cyst bulging into cranial cavity. 5. Dentine exposed, showing through two small apertures where the bone has been absorbed.

from side to side, and weighs 30 grammes, or nearly an ounce. It bears very little resemblance to a tooth, and if situated by itself might have passed as an osteoma. Still, some asperities on its surface show a difference in colour like enamel and dentine alternately. It has a dark patch over its most prominent part, probably due to some antecedent operation.

Careful examination shows that there are at least other two teeth in the cyst, and if there are only two they must be very much larger than those that have been removed. But what seems to be the crowns of two teeth project into the cerebellar cavity, without any covering

of bone, and form the most prominent part of the cyst in that direction.

A beautiful specimen contained in the College Museum is not unlike the one already described. The cyst, if that can properly be called a cyst which bears much more resemblance to a tumour, is on the same side of the skull and in almost exactly the same position. An uneven nodulated mass of dentine extends from within 1·2 cm. (about half an inch) of the median line behind the occipital crest to the post glenoid process of the squamous temporal, a length of 11·8 cm. (about 4 $\frac{3}{8}$ inches. The front of the skull has been removed to show the abnormality, but two projections, like the fangs of molar teeth, extend forwards so far that they must have caused absorption of the parietal bone, if they did not cause it to bulge outwards.

The petrous temporal, with its auditory process, is more behind



FIG. 2.

1. Left condyle of occipital bone. 2. Left styloid process of occipital bone. 3, 3, 3. Projections resembling fangs of teeth. 4. Large mass of dentine. 5. Cut surface showing crusta petrosa, enamel, and dentine. 6. Point projecting far across cranial cavity. 7. Internal wall of right orbit. 8. Left frontal bone. 9. Left supra-orbital foramen.

than below the cyst, the auditory process expanded—broadest—from above downwards almost in line with the face, and its anterior rim, next to the cyst, is very incomplete. The diameter of the mass at its thickest part is 8 cm. (about 3 $\frac{1}{8}$ inches). There is not much displacement of the occipital bone upwards, although it has been absorbed owing to the pressure of the mass, but downwards the bones are all slightly displaced. The cranial cavity, the olfactory fossa, the orbit, in fact the whole right side of the head, is a little lower than the

opposite one. The larger mass, although held in position by the bones, can now, in its dried state, be moved slightly without moving a smaller mass, not one-third as large, which, situated to the inner side and firmly held by the bones, projects so far into the cranial cavity in front of the tentorium cerebelli that it reaches to within 1.5 mm. (about one-sixteenth of an inch) of the middle line of the cranium. This projection resembles considerably the fang of a large tooth, somewhat rounded, but yet irregular; and the other end of this mass is seen showing through, owing to absorption of the bone, between the glenoid cavity of the squamous temporal and the foramen lacerum basis cranii. But there is no part of this cyst, as far as it is exposed, so distinctly tooth-like as the small tooth described in the first one. Yet in removing the bones a small section of the most anterior projection has been sawn off, exposing the structure of a tooth, and showing clearly *crusta petrosa*, enamel, and dentine.

Another specimen (Fig. 3) from the museum, which has evidently been there from the days of the late Professor Dick, is in quite a different position, although also on the right side of the head. It practically fills the superior maxillary sinus and extends beyond it, bulging outwards in all directions. On the outer aspect it extends 11.5 cm. (about $4\frac{1}{4}$ inches) outwards from the middle line of the head, while the maxillary bone and zygomatic ridge have all disappeared over it. On the opposite side the distance of the outer margin of the zygomatic ridge from the middle line of the head is 9.5 cm. (about $3\frac{3}{4}$ inches). Downwards it extends as far as the fang of the third molar, and even farther in the direction of the nasal sinuses, where it has pushed the vomer over to the left side. Upwards the maxillary protuberance has disappeared, and the highest point of it is on a level with the upper extremity of the palatine crest. It has encroached on the orbit to such an extent that there is only 3 cm. (about $1\frac{3}{8}$ inches) between it and the supra orbital process, while the orbit on the opposite side measures 5.8 cm. (about $2\frac{3}{8}$ inches). The supraorbital process is itself thinned, evidently owing to the pressure which must have been exerted on it through the eyeball, so that it measures under 2.5 mm. in thickness, while the left one measures fully 7 mm. On its inferior or oval surface no sixth molar can be seen, while the posterior border of the fifth is merged in the mass. The bone has nearly all disappeared except a narrow plate representing the right palatine, which is pushed 1.4 cm. (over half an inch) to the left, and also downwards into the roof of the mouth, causing great deformity of the posterior nares. The cyst projects across the nares, so that the right one is completely occluded, while when in the fresh condition, with both surfaces covered by membrane, it must have touched the maxillary side of the left naris immediately over the soft palate. An opening that I can only put the tip of my little finger through in the dried state, in the roof of the left posterior naris, and which must have been much smaller in the fresh condition, is the only passage the animal can have had left to breathe through by the nostrils.

The cyst or tumour itself seems to have been constructed on the conglomerate principle, apparently composed of hundreds of aborted misshapen teeth of very varying forms and sizes, but all apparent in one dense heavy mass.

Other six masses of abnormal dental tissue are preserved in the

College Museum, but, as I have no history of them, it is difficult to say whether they should be classified as cysts or neoplasms. Some of them certainly resemble odontomata more than cysts, others look as if they might have been in cysts. The largest one is a dense ivory looking mass weighed nearly 1·9 kilogrammes (about 4 lbs $\frac{1}{2}$ ounce). A second one of much the same character weighs 347 grammes (about 12 $\frac{1}{4}$ ounces). A third, particularly dense specimen, is only a section, the contiguous section being missing; it weighs 319 grammes (about 11 $\frac{1}{4}$ ounces). A fourth, less dense, weighs 170 grammes (about 6 ounces), while the fifth and sixth seem as if they might have been together in one cyst, and weigh 39 grammes (about 1 $\frac{3}{8}$ ounces) and 26·3 grammes (nearly 445 grains) respectively.

The first specimen illustrated (Figs. 1, 1A, and 1B) was sent in to



FIG. 3.

The 2nd and 3rd right upper molars are seen at the right lower border of the photograph, the 4th is missing, while the posterior border of the 5th is merged in the large dental mass, apparently formed by the aggregation of a large number of abnormal teeth.

me last summer by a medical gentleman in the north of England, with a very brief account of the case. The complete unskinned head of a mare was sent with a request that I would make a *post-mortem* examination and report to him.

The history, so far as I have been able to learn it, was as follows:—The animal, a well bred chestnut filly, was two years old off before anything abnormal was observed about her. Then a slight discharge of a dirty foul material from the front and inner aspect of the base of the right ear was observed. Closer examination revealed a sinus from which the discharge escaped, but there was no perceptible swelling and no apparent pain. As it continued to discharge, the veterinary surgeon

in attendance decided to operate. She was a high-spirited, fiery, hot-tempered animal, but was cast, chloroformed, and the operation proceeded with. Several inches of sinus led down to a bony surface indicated by the probe, but deeply seated. This was scraped, and a dilute solution of hydrochloric acid injected, but no solid bone or tooth removed.

However, the operation promised to be successful, as the discharge ceased, and it seemed to have healed up. But in less than three months it broke out again, and this discharge continued intermittently, sometimes ceasing for a couple of months only to again break out. Notwithstanding this, the owner had her trained, as it did not seem to affect her health or spirits; the arrest of the discharge did not cause any perceptible swelling, nor, as far as could be made out in such a fiery, sensitive animal, did it seem to be painful. After she was trained the owner sold her to the country doctor.

The case from which specimen No. 2 (Fig. 2) was obtained is described in this *Journal*, Vol. II. p. 152, by the late Principal Walley, and a brief summary of it here will be instructive.

A cart horse about sixteen years old had been in the possession of the then owner for about eight years without having experienced any distinct illness. Seized with colic one morning, he was treated, seemed relieved from pain, but when seen at 4.30 P.M. looked dull and depressed, and refused his food. His pulse was about sixty, but no other symptom was thought worthy of notice. The following day he appeared much better, but about 6 P.M. the horsekeeper heard him fall, and went and raised him, but did not observe anything to alarm him. About 9 P.M. he was unsteady on his legs, plunged forward, struck his head against the manger, and rolled on to his side.

At 6.30 A.M. on the third day he was lying quietly on his side, semi-conscious. Pulse fifty-six, fairly strong, but occasionally irregular, respirations fifteen, temperature 97.3° F., pupils dilated, eyelids partially closed, twitching movements of the eyeballs, and now and then a convulsive flexion of the head on the body, insensibility to the prick of a pin, but perfect control of the sphincters. At 4 P.M. coma more pronounced, pulse weaker, and mouth partly open. Death took place quietly about 5 A.M. on the fourth day.

The *post-mortem* examination revealed a twist of the colon which had been in existence for some time. The cranial bones were intact, but an abscess occupied the place of the posterior fourth of the right cerebral hemisphere, and a large tumour projected from the squamous temporal bone on the right side, and seemed to have been the cause of the abscess. The contents were unusually fluid, dirty yellow, and composed of softened brain matter.

Now, had we not had the history of this case so clearly set forth, I could not have believed that this horse, with his brain compressed as it must have been, could have worked up to within a week of his death without manifesting any morbid symptoms. It serves to show how much the most delicate structures will stand when the change is sufficiently slow and chronic.

A peculiar case of a dermoid cyst in the centre of the tongue of an ox is related by Prof. Varnell in the *Veterinarian* for 1896, p. 702. The cyst was about $2\frac{1}{2}$ inches long by about $1\frac{1}{4}$ broad, ovoid in shape, its thickest end towards the root of the tongue, and it did not seem

to have any communication with the surface of the organ. It seemed lined by skin, and contained granular matter, hair, epidermal scales, and débris.

Now this cyst, a description of which I have summarised from Prof. Varnell, seems to have been a true dermoid, although it contained neither bones, teeth, nor cartilage. It was deeply seated in a position where normally there would be no hair over it, and no communication with the lingual epithelium could be made out.

But those cysts which are situated subcutaneously, and which are usually classed as dermoid cysts, are certainly not embryomas, as Dr Beard calls them. They have an entirely different origin, and are due to the duplicature or enfolding of the normal skin. They only contain epidermal tissues, hair, sebaceous matter, epidermal or epithelial scales, débris, etc., while teeth, bone, cartilage, etc., are all absent. A minute opening usually connects the cyst with the surface of the body, although, as a rule, they are quite dry and no discharge escapes from the opening of these cysts, the orifice often being occupied by a swirl or tuft of hair, which, however, does not fill, much less plug the opening. In the living animal these cysts sometimes feel quite soft and pillowy, like a cushion. This seems due to evaporation through the opening, and absorption from the cyst being more than sufficient to compensate for the secretion taking place from its walls. These, which we might term cuticular cysts, are innocent harmless structures, compared to the true dermoids which go on growing, and, although not classed as malignant, may cause death owing to their expansion, and displacement of other organs.

As our interest in these dermoids as veterinary surgeons is greatest from the clinical point of view, we have shown that these temporal dentigerous cysts can be operated on with considerable hopes of success, yet the chances should be calculated. In the majority of cases there is a sinus discharging a foul, dirty greyish fluid. This may be harmless in itself, but certainly most horse-owners would like to get rid of it. Then, if the animal is under maturity, which is usually the case, there is the almost positive certainty that the cyst will go on growing, and in growing it may still further invade the walls of the cranium, or even extend into its cavity.

So, notwithstanding the possibility of its harmlessness, the knowledge that it is deep seated, under the scutiform cartilage probably, the risk there is of dangerous hæmorrhage from the possibility of wounding the anterior auricular artery, the possibility of failure to successfully finish the operation, owing to the tooth or teeth being too firmly embedded in the bone, and of a fatal result from injury to the cranium or encephalon, still we think the practitioner is justified in advising operative interference, and the surgeon is justified in operating.

Yet prudence should always be exercised, and if after cutting down on, laying bare, and as far as possible circumscribing the abnormality, it is found that much chisel and mallet or gouge work is required to loosen the dentigerous part of the cyst from its impaction in the bone, the owner should be warned that further procedure, although possibly successful, is certainly at the risk of causing a fatal termination.

Since this paper was written a case has been recorded in this *Journal*, Vol. XV., p. 266, by Mr F. H. Ridler and Prof. Hobday, in which the Professor's former case is referred to.

These two cases, both in mares, one four, the other nine years old, certainly encourage operative interference. Although the older animal required to be operated on twice, and evidently considerable violence used to effect the removal of the offending substance, a favourable recovery is recorded. But there can be nothing more certain than that in the two cases related in this paper operative interference would have had little, if any, chance of being successful.

EXPERIMENTAL DEMONSTRATION OF THE UNITY OF TUBERCULOSIS.¹

By S. ARLOING, LYONS.

TEN years ago there seemed to be general agreement with regard to the unity of tuberculosis in mammals. A few pathologists, in imitation of Virchow, perhaps maintained some idea of keeping separate bovine tuberculosis, the lesions of which appeared to them to be of a special nature. But their tendencies were repressed by the fact that pathogenic properties were similar in human and bovine tuberculosis.

Moreover, it is at least tacitly in favour of this opinion, which is based principally on the presence of Koch's bacilli in all the lesions, and on the experimental production of tuberculosis with infecting products either of human or bovine origin, that steps were everywhere taken to regulate the use of animal products suspected of containing the tuberculous virus.

Doubts with regard to an *absolute* identity were introduced in 1892, with the works of Theobald Smith, R. Gaiser, Frothingham, and Dinwiddie.

Th. Smith, who first took up this question, and Dinwiddie, made the most extensive comparative studies of human and bovine tuberculosis. Smith had been struck by the morphological differences offered by the two bacilli. According to him, the bacilli of bovine origin were short, straight, thick, and of uniform diameter, sometimes swollen in the middle, and strongly resisted decoloration by acids. The bacilli of human origin were thinner, longer, more or less curved, grouped in larger or smaller numbers, and less resistant to decoloration by acids. On solid serum, the culture of the bovine bacillus spread evenly over the nutritive medium, and assumed the appearance of a scarcely visible veil; while that of the human bacillus yielded warty masses, which were dry on the surface.

After some experiments on inoculation, Smith also pointed out some differences in respect of pathogenic properties. The bovine bacillus generally manifested a greater virulence for animals than the human bacillus, a difference observed by Villemin. In spite of this, he concluded, not that there were two distinct species, but that the characters of human and bovine bacilli were variable. According to him, the tubercle bacilli of mammals form a compact group, in which, nevertheless, varieties exist.

Dinwiddie's first work, dated 1899, concerned the question of the relative virulence of human and bovine tuberculosis for the domestic

¹ Translated from the "Journal de Méd. Vétérinaire," May 1903.

animals. He had been inspired by the researches of Smith and Frothingham. His other works are dated 1900-1901.

Dinwiddie found that infective material of bovine origin is more active than that of human origin, that the bacilli present analogous differences, and that bovine tuberculous matter presents a variable activity. He estimates that the lesions peculiar to tuberculosis of the ox in its typical form are due to a particular kind of reaction on the part of the animal, rather than to any special characters which would distinguish bovine from human bacilli. He thinks that the danger of man being infected by cattle has been generally exaggerated. Nevertheless, as the differences which he has observed denote degrees of virulence, rather than different virulences, he thinks it always wise to take measures of precaution, especially with regard to milk.

Frothingham and Gaiser made fewer experiments, from which it appeared that human tuberculosis was unable to propagate in the calf further than at the point of inoculation. But, we repeat, not one of the previous authors dreamt of making two distinct specific groups of the two forms of tuberculosis. On the contrary, this idea occurred to Professor Robert Koch, and he expressed it, under solemn circumstances, at a general meeting of the British Congress on Tuberculosis, in July 1901. In the experiments which led him to this subversive opinion, Koch had for co-worker Professor Schütz, of the Berlin Veterinary School. In support of their proposition, Koch and Schütz have presented arguments of two kinds: some, borrowed from clinical observation, have simply the value of a probability; the others, drawn from experiments, are, on the contrary, of great value, as they can be submitted to a rigorous control.

In this work we will only take up the last named. We will examine the former in a later study.

THE EXPERIMENTAL CRITERION OF KOCH AND SCHÜTZ.

On the ground of thirty-four very expensive experiments, in which the inoculation of human and bovine tuberculosis in large herbivorous or omnivorous animals was comparatively studied, Koch and Schütz affirm that these two forms of tuberculosis are different, *because the human bacilli, contrary to the bacilli of bovine origin, do not infect cattle, whatever the means of introduction may be.*

By cattle, these *savants* mean the ox, sheep, pig, goat, and donkey.

Koch and Schütz call to their aid the results obtained by some previous experimenters, notably Chauveau, Gunther and Harms, Bollinger, Smith, Dinwiddie, and Frothingham.

We have already said that the three last-named authors had studied the comparative inoculation of the bacilli of the two forms of tuberculosis, as well as the evolution and characteristics of the two kinds of bacilli; that they had pointed out differences, and that they had even raised the question of separation before the experimenters of Berlin, but had stopped *en route*.

Koch and Schütz had no such hesitation; but, in affirming that Chauveau, Gunther, and Harms arrived at the conclusions which they expressed in London, they are mistaken. I will not describe the

experiments of M. Chauveau. It will be enough to recall that this experimenter obtained the infection of the ox by feeding with products of human and bovine tuberculosis under its different forms. The conclusions which he drew from this were so clear that, in his opening address at the Congress for the study of tuberculosis, held at Paris in 1888, he did not hesitate to write: "Therefore nothing is lacking in the demonstration of the identity, originally not recognised, of the two forms of tuberculosis. There is only one disease, only one virus, attacking both the bovine and the human species, and capable of being transmitted from the one to the other."

Bollinger, on his side, after numerous experiments made by himself or his pupils, chiefly on the pig, expressed himself several times in favour of identity, notably in 1899, at the Berlin Congress. On this occasion he proclaimed that tuberculosis of cattle and pigs is, from the point of view of etiology, identical with that of man; because of its enormous extension, it is not only a veritable scourge of breeding and agriculture, but also a serious danger for man.

As for Gunther and Harms, they had no particular reasons for figuring in the question raised by Koch and Schütz, for in the interesting experiments that they performed on the contagion of tuberculosis they never took the infecting material from man. This was taken from the ox, save in one case, where it was taken from the monkey.

It will then be seen that Koch and Schütz have not been very happy in the choice of their references. Hence, on them, and on them alone, rests the responsibility for the dualistic theory, by the aid of which they intended to revolutionise the prophylaxis of tuberculosis.

From these few lines we conclude that Koch's opinion rests on the *non-inoculability of human tuberculosis to herbivorous animals or the pig*. Let us see, by the help of our own experiments, whether this text is unimpeachable, and whether it can serve as the foundation of a new idea.

THE AUTHOR'S OWN EXPERIMENTS REGARDING THE INOCULABILITY OF HUMAN TUBERCULOSIS TO LARGE ANIMALS.

We have successfully inoculated the bacillus of human tuberculosis to the donkey, the goat, the sheep, the ox, and the pig. Let us briefly describe these inoculations.

(a) *Inoculation of Human Tuberculosis to the Ass*.—The experiments on this point go back to 1896.

Some particles were taken from the surface of a culture of human bacilli on glycerinised potato, and made into an emulsion with water. These bacilli had been isolated from the lesions in a guinea-pig; that is to say, they infected this animal very easily.

With the emulsion, previously filtered through sterilised linen, two donkeys were inoculated in the jugular vein. One of the donkeys, which was not very strong, rapidly became weak, and died at the end of twenty-eight days.

The other, young and vigorous, continued to appear healthy. However, the normal temperature oscillated in the course of the first

month, and rose sometimes to 1° above normal. It was killed at the end of two months.

The *post-mortem* revealed tuberculous lesions in the lungs of both subjects. In the first, congestive lesions were found here and there, and everywhere little tubercles which felt firm to the finger. In the second, in passing the finger over the surface of the lungs, one felt a large number of circumscribed points where the consistence of the tissue was increased. These corresponded to tuberculous nodules in a state of retrogression, as was proved by the histological examination. The microscope has likewise shown that the nodules in the lungs of the first donkey were tubercles in process of formation.

In another experiment two intravenous inoculations were made at intervals of a fortnight. The temperature oscillated after each injection. The maximum was 1° in the first period, and 1.5° in the second. When tested with tuberculin after the second injection, the donkey had an elevation of temperature of 2.9° C. It was killed two months from the beginning of the experiment.

The *post-mortem* showed a great number of little elevations in the lungs, often surrounded by a little ring of congestion; and a great number of round whitish spots of the diameter of a lentil. The histological examination showed that the bright red points were tuberculous granulations, isolated or close together, and in full activity; while the whitish spots corresponded with tuberculous granulations of which the cellular elements were more or less compressed by the connective-tissue fibrils.

Hence, certain pure cultures of human bacilli are capable of infecting the donkey when they are introduced into a vein. Hence, also, the donkey can recover spontaneously from pulmonary tuberculosis produced by an intravenous injection of human bacilli, a remark already made by M. Chauveau.¹

(b) *Inoculation of Goats with Human Tuberculosis*.—The experiments described in this paragraph were commenced 20th April 1899. At this date, seven goats received into the jugular vein an injection of an emulsion of Koch's bacilli of human origin, taken from a culture on glycerinised potato. Three goats had not previously been the subject of any experiment. Four had been treated for a long time with substances which have a certain reputation in the therapeutics of tuberculosis. Two of the goats previously untreated succumbed in less than a month. The five others were killed on the 8th July of the same year. The goats which died quickly grew thin, coughed, and experienced difficulty in respiration almost immediately; their temperature was frequently above 40° C., and even went up to 41.3° C.

The *post-mortem* showed considerable lesions in the lungs, all consisting of tuberculous granulations, as was seen from the histological examination.

All the others showed signs of infection in different degree—elevation of temperature a few days after the injection, cough after the eighth or twelfth day, and emaciation. During the last month their temperature oscillated between 39.1° and 39.7° C. At the *post-mortem* the lungs presented a tuberculous eruption. This eruption was composed of

¹ These experiments were published in the *Journal de Physiologie et de Pathologie générale*, for July 1900.

granulations of which the largest were of the diameter of a millet-seed. The granulations were confluent on the anterior lobes of the lungs. The lymphatic glands at the entrance to the chest, and those in the mediastinum near the œsophagus, were more or less swollen. Some of the granulations showed commencing caseous degeneration towards their centres.¹

(c) *Inoculation of Human Tuberculosis to the Ox, Sheep, and Goat.*—In this paragraph five series of experiments made after the communication of Koch and Schütz in London will be described.

Each series comprised one or two young bovine animals, one or two sheep, and one or two goats. All these animals were recognised as being free from tuberculosis. Tuberculin was used according to directions in the case of the bovine animals.

In addition to the proper subjects of each series, we used a few rabbits and guinea-pigs in order to obtain information with regard to the virulence of the infecting material used in each experiment. This infecting material consisted of human bacilli cultivated on glycerinised potato. For each series human bacilli taken from different patients were used, so that our experiments are not the repetition of a simple test; in this respect they differ from those of Koch and some other experimenters. We used different bacilli because we knew the extent to which tubercle bacilli can undergo modifications from the pathogenic point of view.

The bacilli were made into an emulsion with water containing 7 per cent. of common salt. The emulsion was made in fixed proportions, generally one part of bacilli to twenty-five of water, then filtered through a fine linen sieve, and finally injected into the jugular vein or an auricular vein. The intravenous injection has been almost uniformly adopted so as to render comparison of experiments easier, and infection more certain.

First Series.—Emulsion of 1 in 25 of a human bacillus isolated from the sputum of a patient, and cultivated on potato; of medium activity for the rabbit and guinea-pig. With this the following injections were made:—

Yearling Heifer	4 cc.
Calf fed on milk	2 cc.
Two sheep	2 cc.
One kid	1·5 cc.

One sheep and the kid died tuberculous in thirty-seven days. The calf died accidentally at the end of thirty-two days, but after having wasted away. The other sheep and the heifer were killed, the former at the end of twenty-five days, the latter after 120 days. Discrete tubercles in the lungs of the heifer; exquisite eruption of tuberculous granulations in the calf and the sheep; more diffuse and extensive lesions in the kid.

Otherwise expressed, complete success in all subjects; moreover, verified by histological examination.

Second Series.—Emulsion of 1 in 25 of bacilli obtained from a case of tuberculous pleurisy, which subsequently recovered. These bacilli were passed twice through the guinea-pig and afterwards

¹ These experiments on the goat and ass enabled me, in the next issue of the *Revue de la Tuberculose* (August 1901), to dissent from the view expressed by Koch in London.

cultivated on glycerinised potato and solid blood. The following were inoculated with the emulsion :—

One weakly calf	2 cc.
Two sheep each	1·5 cc.
One goat	1·5 cc.

The calf died of tuberculosis at the end of seventeen days ; the sheep and the goat recovered after having presented signs of infection, and were killed eighty days after inoculation.

At the *post-mortem* a great number of nascent tubercles were found in the calf ; in the other subjects a tuberculous infiltration of the lungs and of the pulmonary pleura, not so considerable, however, in the goat as in the sheep.

Microscopic examination showed that the liver and spleen of the sheep and goat were affected with tuberculosis.

Third Series.—Bacilli obtained from a fatal case of tuberculosis involving the pleura, pericardium, and lungs ; cultivated in the same manner as those in the preceding series. With the 1 in 25 emulsion the following were inoculated :—

One young bull	3 cc.
Two sheep each	1 cc.
One large goat	2 cc.

The bull, previously healthy, died of tuberculosis in thirty-two days, having shown frightful difficulty in respiration. One sheep died accidentally. The other sheep and the goat were killed forty-two days after inoculation.

In the young bull the lungs were crammed with fine tubercles surrounded by inflammation ; the bronchial and mediastinal glands were enormous. Liver yellowish and fatty in appearance.

A great number of small sub-pleural and intra-parenchymatous tubercles in the sheep's lungs ; smaller in the goat. The microscope revealed a generalised fatty degeneration of the liver, and a great number of small young tubercles, often situated within the lobules.

Fourth Series.—Emulsion, 1 in 25, of bacilli obtained from the sputum of a tuberculous patient, and cultivated in a state of purity on glycerinised potato. With this the following were inoculated :—

One heifer	5 cc.
One sheep	2 cc.
One sheep	2 cc. in the peritoneum.

The emulsion was diluted to 1 : 500, and then used to inject into the jugular of

One calf	2 cc.
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The calves, except for a slight cough, appeared to be healthy ; however tuberculin caused a marked reaction. They were killed at the end of 180 days. The sheep also appeared healthy, but they reacted to tuberculin. They were killed ninety days after inoculation.

The lungs of the heifer appeared to be healthy ; those of the sheep led one to suspect the presence of very minute sub-pleural tubercles. That is to say, a macroscopic examination seemed to indicate a complete failure. But with the microscope very fine tubercles could be

seen in the pulmonary parenchyma, principally along the bronchial tubes. Failure was therefore only apparent, and the bacilli used for inoculation were not inactive. Moreover, the sheep inoculated in the peritoneum proves this, for in this animal the glands scattered in the furrows of the stomach were found to be hypertrophied and tuberculous. Their centre was transformed into a caseous mass, which had assumed the aspect of analogous lesions in bovine tuberculosis.

Fifth Series.—Emulsion, 1 in 25, prepared with cultures of a bacillus found in the glands of a man affected with dropsy. With this emulsion the following were inoculated in the jugular—

One calf	4 cc.
One sheep	2 cc.
One goat	2 cc.

These animals grew slightly thinner during the fortnight following inoculation, and then gradually regained their former condition. However, they all reacted distinctly to tuberculin.

An inoculation similar to the first was made. The animals resisted admirably. They were killed between the 120th and 150th day from the commencement of the experiment.

At the *post-mortem* the lungs appeared healthy. Very careful observations had to be made to find signs of subpleural lesions. However, the calf showed hypertrophy of several lymphatic glands. A pulp made from these glands caused tuberculosis in guinea-pigs. The hypertrophy was therefore of a tuberculous nature.

But, although the lungs appeared healthy to the naked eye, they contained fine lesions which were visible with the microscope. Nodular infiltrations were found in the liver and kidneys.

The five series of experiments described above may be divided into two groups. The first group includes the inoculations of the first, second, and third series. The three strains of human bacilli used in these experiments gave results which were undoubtedly positive. They caused pulmonary lesions which were numerous and obvious; moreover, microscopic tuberculous lesions were present in the liver, spleen, and kidneys, bearing witness to the generalisation of the tuberculous process.

The second group includes the fourth and fifth series. The bacilli used in these experiments only caused very slight lesions, which might have passed undetected in a superficial examination or by any person desiring a negative result. But the tuberculous lesions were manifest in the microscopic sections of the lung.

Bacilli of human origin can therefore infect the ox, sheep, and goat. However, it must be added that their virulence has presented various degrees.

(d) *Inoculation of a Pig with Human Tuberculosis.*—An emulsion of 1 in 25 was made with the bacilli used for inoculation in the first series of experiments just described, and 4 cc. of this emulsion were injected into the auricular vein of three young pigs.

From the following day the three subjects appeared to be ill. Two remained lying down, and refused all food. Their condition became worse during the following days. A month and a half after inoculation it seemed that one of the pigs must shortly die, and it was

slaughtered. On the contrary, the condition of the other two pigs improved; they were kept for thirteen months, growing and fattening like perfectly healthy subjects. They were killed.

The pig killed at the end of a month and a half presented some patches of pleurisy. At this level the pleura presented some large more or less projecting tubercles.

The bronchial and mediastinal glands were voluminous and tuberculous. The malpighian bodies of the spleen were hypertrophied, and some were tubercular.

The microscope confirmed all these lesions, and, further, allowed tuberculous alterations to be observed in the spleen, the liver, and the kidneys.

One of the subjects kept for thirteen months, fat and in good condition, presented here and there in the lungs tuberculous masses which were caseous in the centre, and varied between the size of a lentil and that of a haricot bean. The bronchial, mediastinal, and retro-pharyngeal glands, and a few mesenteric glands, were hypertrophied, and contained caseous tubercles. In the liver a few caseous nodules were found; in the spleen one was found near one of the extremities. The other pig, in very good condition, appeared at the first glance to have perfectly healthy lungs; but after slight dessication a few very small, whitish, pleural granulations were perceived. The bronchial, mediastinal, retro-pharyngeal, and mesenteric glands were tumefied and hard, and showed on section some sclerotic points, like old tubercles in the fibroid state.

To sum up, in all the three pigs the success of the experiment was complete; but in course of time the tuberculous process became circumscribed, and, according to the subject, the lesions underwent either caseous degeneration or fibroid transformation. The results in these experiments have been influenced by the predisposition or resistance peculiar to each subject.

(e) *Inoculation of the Ox, Sheep, and Goat with Bovine Tuberculosis.*—It seemed indispensable that we too should compare the human bacilli with those of the ox in the matter of virulence. To this end we carried out three series of experiments similar to those described in paragraph c.

First Series.—Bacilli obtained from a tuberculous lesion in an ox, and grown as a pure culture on glycerinised potato. An emulsion of 1 in 25 was injected into the jugular vein of—

One calf	.	.	.	2.5 cc.
One large goat	.	.	.	1.5 cc.
One sheep	.	.	.	1 cc.

The calf very quickly became ill, presenting signs of very grave acute pulmonary tuberculosis; it died twenty-five days after inoculation. The sheep followed suit, and died on the forty-fourth day. As for the goat, after having also shown alarming symptoms, it partially recovered. It was killed at the end of 150 days, being then in a rather sorry condition.

The lungs of the calf were everywhere infiltrated with tuberculous granulations surrounded by inflammatory lesions. Enormous bronchial and mediastinal glands, recalling those of the calf in series 3 of inoculations with human tuberculosis. The retro-pharyngeal glands

were hypertrophied, but without apparent tuberculous lesions; nevertheless, they contained bacilli, as was proved by the inoculation of a guinea-pig with pulp from them.

The lungs of the sheep were greatly distended by a generalised infiltration, in which the eye could easily discern a great number of tubercles. The spleen was hypertrophied, the follicles being more distinctly visible than usual. Some very fine granulations had formed in the liver.

The lungs of the goat were dotted, both under the pleura and in the depth of the parenchyma, with large grey tubercles without any peripheral inflammatory zone; some parts of the anterior lobes were hepatised. Many tuberculous granulations in the liver. Very few nodules in the spleen. The histological examination confirmed the results of the *post-mortem*.

Second Series.—Another bacillus of bovine origin, cultivated on glycerinised potato; emulsion, 1 in 25, inoculated into the following animals:—

One calf	2'5 cc.
One goat	1'5 cc.
One sheep	1 cc.

From the fourth day the calf began to cough; from the tenth the three animals suffered greatly—cough, rapid breathing, and fever.

The sheep died at the end of twelve days, the goat at the end of seventeen days, and the calf at the end of eighteen days.

Confluent eruption of fine tubercles in the lungs of the calf, causing here and there complete hepatisation. Bronchial and mediastinal glands hypertrophied and pulpy. In the sheep and the goat the pulmonary lesions were similar to those described in the calf. The sheep was more gravely attacked than the goat.

A second calf was inoculated with the same bacilli, only the emulsion was 1 in 200 instead of 1 in 25. Little change was remarked in the animal during ten days, but suddenly it became very ill; it was killed when in a dying condition on the fiftieth day.

Tubercles were scattered in the lungs, larger but less confluent than those in the lungs of the calf which died soon after inoculation. In some parts of the right lung the tubercles were surrounded by recently-formed inflammatory lesions, rapidly approaching hepatisation.

Hence, a very weak dose of these bacilli can bring about fatal results.

Third Series.—In this series, the infecting material was taken from pleural tubercles in an ox, made into a pulp, diluted, and filtered. The following inoculations were made with it:—

One small heifer	2'5 cc.
One goat	1'5 cc.
One sheep	1 cc.

The heifer showed characteristic symptoms, and died twenty days after inoculation. The goat became rapidly ill, and died on the twenty-second day. The sheep died on the thirty-first day.

The lungs of the heifer were riddled with tubercles embedded in

inflammatory lesions, which at some places rendered the tissue solid ; the parenchymatous organs were congested and swollen, but not apparently tuberculous.

In the goat, the peri-tuberculous inflammation gave to the lungs an appearance of a greyish block, the fragments of which have fallen to the bottom of the water. The liver presented small tuberculous lesions. The lesions were almost identical in the sheep.

The matter taken from pleural tuberculous "grapes" of the ox thus acted in the same way as the pure culture of bovine bacilli used in the two preceding series.

(f) *Inoculation in the ox, sheep, and goat, with equine tuberculosis.*—In order to complete our comparative study, we made a series of inoculations with bacilli of equine origin cultivated on glycerinised potato. With an emulsion of 1 in 25 we inoculated into the jugular :—

One heifer	2.5 cc.
One goat	1.2 cc.
One sheep	1 cc.

The goat died from an intervening disease. The heifer having grown very thin, it was killed at the end of sixty-seven days. The sheep offered a greater resistance to infection ; it was killed on the sixty-fifth day.

In the lungs of the heifer there were granulations and small tuberculous masses, of the size of a lentil or small pea, and without an inflammatory zone. The centres showed a tendency to degeneration. The bronchial and mediastinal glands were infiltrated with a yellowish tuberculous matter. The lungs and the glands of the sheep presented similar lesions. The goat, having died prematurely, presented no very evident lesions. Nevertheless, very small sub-pleural swellings and a few fine granulations were observed in the pulmonary parenchyma.

(g) *General Examination of the Author's own Experiments:*—The inoculations described in the preceding paragraphs are forty-six in number—thirty-three were made with human bacilli, and thirteen were made with animal bacilli.

The human bacilli were taken from five different sources, the animal bacilli from four sources—three from the ox, one from the horse. The thirty-three animals devoted to inoculation with human bacilli were taken from the different species which provided subjects for the experiments carried out by Koch and Schütz. It was the same with the thirteen animals used for inoculation with bacilli of animal origin.

The results were positive in every experiment.

The results of the experiments relating to inoculation with bovine tuberculosis will surprise no one. But Koch and Schütz, as well as the partisans of their doctrine of dualism, will perhaps be astonished at the success of the inoculations made with the human bacilli. It is therefore well to insist on this point.

The five bacilli of human origin which we have studied have all infected the ox, sheep, goat, donkey, and pig by intravenous injection, and caused more or less extensive tuberculous lesions in all the parenchymatous viscera.

However, we must add that the virulence, as measured by pathogenic

aptitude and toxic properties, was not the same in all. Three of them did not cause fatal tuberculosis in any of the animals submitted to the experiment, and two of the latter only showed microscopic lesions in certain species, namely the bovine species. But, thanks to histological examinations, and to the presence of subjects of several species in the series, one may be assured that the two bacilli were not entirely lacking in pathogenic power.

However, if there are human bacilli which are only slightly pathogenic for animals, there are others which are very active and of which the pathogenic power may be compared with that of the majority of bacilli of bovine origin. Thus, one of ours, that of the second series, caused fatal tuberculosis in the ox and sheep; another, that of the third series, killed the ox, sheep, and goat, just like the bacilli of bovine origin, and in as short a lapse of time.

We must add, to be complete, that, as a general rule, bovine tuberculosis has shown itself to be less infective. Whereas almost all the subjects inoculated with bovine tuberculosis died from the infection, and not later than forty-four days afterwards, the majority of those inoculated with human bacilli had to be killed. But the subjects killed by the infection were not scattered about in the different series; they belonged to particular series, from which we must conclude that among the bacilli from different sources some are very virulent.

It is therefore impossible for us, after our experiments, to accept the criterion laid down by Koch and Schütz for separating human tuberculosis from bovine. On the question of the duality of tuberculosis, we therefore entirely disagree with the German *savants*.¹

CRITICISM OF THE VALUE OF NEGATIVE AND OF POSITIVE RESULTS. INTERPRETATION OF RESULTS.

Before finishing, our duty is to look for the cause of the divergencies in the results obtained by Koch and Schütz and those obtained by the majority of the other experimenters who have studied this question.

We admit that Koch and Schütz may have failed to transmit human tuberculosis to large herbivorous animals, although we make reservations with regard to several experiments which they judged as negative, either because they employed an entirely personal method of estimation, or because they did not look for lesions in microscopic sections and in all the parenchymatous viscera.² But these negative results, if they existed, can avail nothing against the positive results obtained on all hands. Nevertheless, in order to work scientifically, we must look for the determining factor, and see if it cannot be reconciled with the dogma of the identity of human and bovine tuberculosis.

To us, the negative, or quasi-negative, results denote the feeble virulence of the bacilli used in inoculation; while the more or less serious positive results, sometimes equal to those following an inoculation with bovine tuberculosis, are due to bacilli which are more virulent.

In our article which appeared in the *Revue de la Tuberculose* for

¹ Immediately after Koch's lecture at the Congress in London, Lister, Nocard, M'Fadyean, Thomassen, and Ravenel expressed disagreement with the lecturer, or made strong reservations with regard to his position.

² We dealt with this point in our communication to the "Académie de Médecine," on the 24th Dec. 1901.

August 1901, we said on this subject: "I explain the pathogenic differences observed in the experiments of Koch and Schütz by the variations in the virulence of the bacillus, and by the particular degree of susceptibility possessed by certain species of animals, and even by individuals."

And in December of the same year, in a communication to the *Académie de Médecine*, we said: "As for the tuberculous virus, Koch and Schütz reason as if it had a fixed and invariable activity. Koch admitted this fixity in his first works on tuberculosis. Since then it has been shown that the bacillus of tuberculosis, like many other microbes, presents frequent variations *in vivo* and *in vitro*, and I consider it an honour to have contributed to the establishment of this opinion, either directly or through some of my pupils," several of whom are now teachers.

In short, we believe we were perhaps the first in 1884 and 1886 to fix attention on the *attenuation* of the tuberculous virus, on the occasion of an experimental study of the tuberculous lesions called scrofulous, and of visceral tuberculosis.¹

The differences which we have pointed out between the forms of tuberculosis have been recognised as exact; but the explanation which we gave has not been readily accepted. Instead of allowing, as we do, that it depends on the difference of the activity of the virus, Eve, Straus, de Renzi, Leloir, and Nocard preferred to put it down to the *number* of bacilli present in the lesions.² At first these authors did not believe, and some never have believed, in a real attenuation of the bacilli.

In spite of the objections which have been raised, we have continued our comparative studies, which embraced the diverse manifestations of surgical tuberculosis (tuberculosis of bones and joints). Among these forms of surgical tuberculosis we have found the tuberculous virus with several degrees of activity, using as test the susceptibility of the rabbit and the guinea-pig.³

A little later, in conjunction with Courmont, we examined lupus, and we proved that the virus contained in the lesions of cutaneous tuberculosis designated by this name was far from being always of the same activity.⁴ Finally, at my instigation, Courmont and Denis studied the degree of virulence in several cases of pulmonary tuberculosis of which the clinical symptoms were different, and there again we were able to observe that certain cases were associated with very virulent bacilli, and others with attenuated bacilli.⁵

We have published some experiments with the object of showing that the weakened pathogenic properties of certain tubercular viruses depend more on the attenuation than on the diminution of the number of the bacilli. We have gathered together all the facts in our *Leçons sur la tuberculose*⁶ pp. 46 and following. Nevertheless, we have not yet succeeded in convincing Leloir, Straus, and Nocard, as is easily seen from what took place at the Congress for the study of tuberculosis held at Paris in 1892, and in reading Straus's book, *La tuberculose et son bacille*, pp. 788 and following.

¹ See Arloing: "Compte rendu de l'Académie des Sciences," 1884 and 1886. Also "Revue de Médecine," 1887.

² See Congress for the study of Tuberculosis, 1886, p. 41.

³ See Congress for the study of Tuberculosis, 1899, p. 404.

⁴ See Arloing and Courmont: Congress de la Tuberculose, 1893.

⁵ See Courmont and Denis: "Revue de la Tuberculose," 1894.

⁶ See Arloing: "Leçons sur la Tuberculose," Paris 1892.

From our earlier studies we were therefore prepared to take an interest in the conflict raised by Koch, and to attempt to solve it by the variability of the bacillus of tuberculosis. The variability of the bacillus in its manner of vegetation, in its form, in its virulence, exists in bovine tuberculosis quite as well as in human tuberculosis, with this restriction, which it seems prudent to make at present, that the most attenuated varieties of the bovine bacillus have a higher virulence than the average human bacilli. So that we imagine the bacilli of human and animal origin as forming a scale of virulence, with numerous and undetermined limits. Now, these varieties were formed in some way unknown to us, by the action of living and inert media, and some are adapted so exactly to certain organisms that they seem to have changed their properties when they are suddenly transferred to other species of animals. But prolonged observations in the domain of tuberculosis permitted of the discovery of the phylogeny of these varieties, which no one would be inclined to regard as different species.

To come back to the question which we had put to ourselves, we will say that Koch and Schütz have experimented with two varieties, one human, the other bovine, as different as possible from one another in virulence. Hence the results, rather superficially gathered together, which caused them to admit a fundamental difference between the two varieties. If they had tried several bacilli of different origin, they would probably have met with intervening virulences which would have prevented them from digging a veritable trench between human and bovine tuberculosis.

In that respect, our experiments differ from theirs, and offer a peculiar interest.

The explanation which we offer to-day, renewed from that of 1901 and my communication made last year at the Conference in Berlin, with a view to conciliation, is now generally accepted, except by Koch.

A great variation in the virulence of the bacillus of tuberculosis is readily admitted. This proves that the question has advanced since 1892, at which date we were not agreed to profess belief in the variability of Koch's bacillus. We have therefore felt great satisfaction in seeing our opinion confirmed by the works of Th. Smith, Dinwiddie, de Jong, Ravel, Vøgedes, Veszpremy, von Behring and his pupils, and by the communications of our learned colleague M. Nocard, whom we formerly regretted to see amongst our adversaries. And we begin to hope that when Koch and Schütz shall be pleased to experiment under the inspiration of this opinion, these divergences will cease.

CONCLUSIONS.

From the facts contained in the above article we draw the following conclusions:—

1. Human tuberculosis can be readily inoculated into the ox, and sometimes and by certain ways of inoculation, it causes the lesions characteristic of bovine tuberculosis.
2. The human bacillus is not always of the same virulence, and a given bacillus does not manifest the same degree of activity on herbivorous animals of different species.
3. In some cases, the human bacillus is as virulent as a bacillus of

bovine origin ; in others, on the contrary, its virulence is so much weakened as to appear null, particularly if used on bovine subjects.

4. Human bacilli of attenuated virulence always cause, at least in the lung, after intravenous injections, lesions visible to the microscope, which sometimes tend to rapid fibrous transformation.

5. It is therefore impossible to give an opinion on the failure of an inoculation without a microscopic study of the lung and of the principal parenchymatous viscera.

6. The variability of the virulence of the bacillus accounts for the apparently negative results which led Koch and Schütz to believe in duality.

7. The identity of human and bovine tuberculosis ought to be maintained, and the prophylactic measures which result from it ought also to be maintained, notably with regard to the use of milk.

INTUSSUSCEPTIONS.

By EDREDD M. CORNER, B.Sc. (London), M.A., M.B., B.C. (Cambridge), F.R.C.S. (England), Demonstrator of Anatomy, late Resident Assistant Surgeon, St. Thomas' Hospital.

DURING the years 1901 and 1902 a number of cases of intussusception passed through my hands, and from observations that were made upon them I became convinced that the present state of our knowledge of the pathology of this condition was erroneous. In a paper in the St. Thomas' Hospital Reports, 1901, I raised this question, embodying also my reasons for the attitude taken up and the reports of five clinically interesting cases. If the histories of the sister sciences of anatomy, physiology, etc., are examined, the fact at once becomes patent that although the knowledge gained from mere dissection was continually increasing, no advance of importance was made until the introduction of the comparative method. This comparative method has been used in physiology, medicine, and pathology, and to a less extent in surgery. But observers are much hampered here by reason of their lack of knowledge of the physiological limits of the animals experimented on. Veterinary surgery and medicine have only of recent years been pursued with true scientific spirit, and in this respect are still far behind their corresponding human sciences. As a result, they are as yet scarcely ripe for accurate comparisons ; yet, on account of the larger size of the parts that may be observed, and the still greater frequency of *post-mortem* observations, a great deal may be learned.

In 1898 I made use of this method in an enquiry into the influence of the quadrupedal and bipedal positions on fractures and dislocations in man and animals.¹ The interest of such a comparison has caused me to examine the records of intussusception in animals to see how they shed light upon the pathology. The knowledge gained from this enquiry forms the subject matter of this paper, and I have added a short introductory account of the present views on the pathology of intussusceptions and the alterations that I have suggested.

Before proceeding, I would like to offer my thanks to Prof. Hobday for his scientific interest in my work and the readiness with which he placed his knowledge at my disposal.

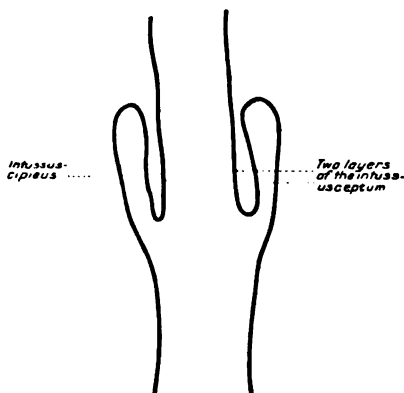
¹ "Lancet," 1898 and 1899, "Veterinarian," 1899.

INTUSSUSCEPTIONS IN MAN.

By intussusception is meant the inversion of a piece of bowel into the part immediately below it, the former being called the intussusceptum and the latter the intussusciens or receiving layer. The invaginated portion then acts like a foreign body to the part of the bowel that receives it, and is consequently passed on by peristalsis. The invaginated portion or intussusceptum naturally consists of two layers, which it is needless to distinguish with names. Thus every intussusception consists of two parts, the intussusceptum and the intussusciens.

The invagination may occur in any part of the alimentary tract except the œsophagus and stomach. When it originates in the small intestine it is termed enteric, and is most common in the ileum, then in the jejunum, and very rare in the duodenum.

The most favourite site for intussusceptions to start at is about the ileocæcal valve. One variety, said to be far the most common in man, is when the ileocæcal valve is pushed into the cæcum, and so forms the head or leading part of the invaginated portion or intussusceptum. This variety is called the ileocæcal.



Another form, which is said to be rare in man, starts in the ileum just above the valve, through which it becomes prolapsed. As it originates in the small intestine, it is primarily enteric, and its mere prolapse through the valve in no way constitutes a second intussusception. This form may be recognised as a subvariety of the enteric, and is called ileocolic. Occasionally the inversion begins in the cæcum or colon; the intussusception is then called colic.

All these are examples of single intussusceptions, *i.e.*, there is only one invagination; and there are four kinds, enteric and its subvariety ileocolic, ileocæcal, and colic.

Human pathologists recognise that sometimes a second invagination may be imposed on the first, rendering it an example of a double intussusception. But so little is said and known of these that it is unnecessary to say more to introduce the subject. The superposition of a third invagination, making a triple intussusception, is known, but is extremely rare.

The accepted views of the relative frequency of these varieties varies within wide limits, a point which in itself expresses the instability of the present ideas of its pathology.

Table showing the Present Views on the Relative Frequency of the Different Varieties of Intussusception.

<i>Author.</i>	<i>Enteric. Percentage.</i>	<i>Ileocolic. Percentage.</i>	<i>Ileocæcal. Percentage.</i>	<i>Colic. Percentage.</i>	<i>Double Intus- suspension, etc. Percentage.</i>
Treves ¹ . . .	30	8	44	9	9
Sargent ² . . .	11	5	69	5	10
M'Adam Eccles ³ .	4	2	87	5	2
Limits of variation	4-30	2-8	44-87	5-9	2-10

The only point on which these figures agree is that over 90 per cent. of cases are examples of single intussusception, and that the ileocæcal variety is by far the most common, whilst the ileocolic is by far the most uncommon. Omitting from consideration such varieties as invagination of the appendix, or a Meckel's divesticulum, the above may be taken as the view accepted by human pathologists. Thirteen successive cases of which I made particular notes, showed that 10, or 77 per cent., were double intussusceptions, and that 3, or 23 per cent., were single! Either I have run across a most remarkable series, or former observers have either from lack of time or scanty examination arrived at a wrong diagnosis. The latter suggestion is recommended by the following considerations:—

1. The reduction of an intussusception is the operation in surgery where speed is most requisite, on account of the tender ages of the patients, etc.

2. I have often seen intussusceptions reduced and returned to the abdomen without examination, and finally called ileocæcal.

3. This variety of intussusception has been diagnosed over and over again in cases in which reduction has been accomplished by inflation, injection of water, or manipulation, and where there has been no autopsy in unreduced cases.

4. Examination *per rectum* reveals the fact that the apex of the invaginated part or intussusceptum feels like the ileocæcal valve. In reality I believe this to be merely the false impression that may be given by the œdematous intussusceptum of any part of the gut.

Similar points might be multiplied, but sufficient has been said to show that the great frequency of single and ileocæcal intussusceptions may largely depend on the lack of observation of the surgeon, or negative evidence rather than positive.

To the truth of the statement just made, as to the conditions of the invaginations in my thirteen cases, I am prepared to adhere, and the chief points upon which I have based my opinion may be briefly given. For a fuller account I would refer to my paper in the *St. Thomas' Hospital Reports*, 1901, XXX., p. 355.

1. A great deal of the invagination is reduced before any small bowel is expressed from the cæcum; a fact suggests that the colic part of the invagination alone has been reduced.

¹ Treves, "Intestinal Obstruction."

² Sargent, "St. Thomas' Hospital Report," 1900.

³ Compiled from three papers in the "St. Bartholomew's Hospital Reports."

2. The difficulty of reducing the last part of the intussusception is due to the prolapse of the ileum through the ileocæcal valve.

3. If traction on the small bowel is not used, a little enteric intussusception may be often expressed from the cæcum.

4. A lateral dimple frequently seen on the side of the ileum after reduction, just above the valve, may represent the origin of the invagination.

5. Examination after reduction reveals the fact that the last half-inch of the ileum is of a much darker colour than the contiguous bowel, and the limits of the discolouration are fairly sharp. Such an appearance suggests that this terminal half-inch has been strangulated, such as through the ileocæcal valve. This is more frequently to be noticed when traction has been employed to reduce the last part of the invagination, whilst if expression only is used a small enteric intussusception or lateral dimple also is seen. The real anatomy of the parts may be easily hidden during the reduction unless a careful watch is kept.

Thus, allowing for the fact that a man's observations may be warped by his own personal equation, there seems ample evidence for the fact that double intussusceptions are of far more frequent occurrence than is generally supposed. And, on the other hand, there are reasons to doubt the accuracy of the diagnosis in a large number of cases reported or recorded as single, and especially those regarded as ileocæcal, whose diagnosis really rests more on negative than positive evidence.

The veterinary literature was examined as thoroughly as possible for recorded examples, and over twenty-five were found. To these may be added four specimens that Professor Hobday has allowed me to see. The first fact that becomes patent is that observers have not recognised what are known humanly speaking as "intussusceptions of the dying," and which occur during the death agony. The characters by which they may be recognised are: (a) their multiplicity; (b) they are easily reduced; (c) they are frequently ascending, *i.e.*, reversed peristalsis; (d) there are no signs of inflammation or change about them; (e) always in the small bowel, etc.

It is thus impossible to say whether the intussusceptions of the dying are a distinctive character of human pathology or not. Some of the features described as occurring in dogs' intussusceptions seem to possess similar characteristics.

NOTES OF THE RECORDS OF VETERINARY CASES.

1. Williams. *Principles and Practice of Veterinary Medicine*, p. 675. "Intussusceptions may involve both large and small bowel in all the domesticated animals." He bears out what I have suggested for man, namely, that the origin is either in the large or small intestine and not at the ileocæcal valve. He mentions a cow who passed the intussusceptum as a slough on the seventh day of the illness, and mentions Aitken of Dalkeith who records a similar event in a cow on the fifth day. The anatomy of the slough is not given in either case, and so there is no suggestion as to the possible variety for either case.

2. E. M. Jarvis. *Journal of Comparative Pathology*, 1894, p. 380.

Mare, twelve years old, suffered from colic, etc. Death in forty-eight hours. *Post-mortem*—The whole of the cæcum was invaginated into the colon. Worms were present in both stomach and small gut. Variety, colic.

3. Penberthy. *Journal of Comparative Pathology*, 1896, p. 48.
Gelding, five years, colic for a week before seen, death on the sixteenth day. *Post-mortem*—"Complete intussusception of the cæcum into the commencing portion of the colon." There was a large cyst containing three pints of fluid in connection with the cæcum. Variety, colic.
4. M'Fadyean. *Journal of Comparative Pathology*, Vol. XV., Part II., 1902, p. 154.
Mare, seven years old, severe abdominal pain. *Post-mortem*—Intussusception beginning in the ileum above the ileocæcal valve, and 3 feet were prolapsed into the cæcum, whilst the ring marking the point of junction of the invaginated and receiving layers was 6 inches above the ileocæcal junction. Variety ileocolic (?). Had been ileocolic-colic.
5. Dewar. *Veterinarian*, 1895, LXVIII., p. 369.
Intussusception "is a well-known although not very common cause of abdominal colic," "best known in the dog," "comparatively common in the ox and more rare in the horse." In the horse it is said to be most common that the ileum passes into the cæcum—that is, ileocolic variety. The reported cases do not bear out this statement.
- Filly, one year old. *Post-mortem*—Double intussusception of the ileocolic-colic variety.
6. Power. *Veterinarian*, 1897, LXX., p. 648.
Two enteric intussusceptions in dogs dosed with turpeth mineral. Cats apparently tolerate intussusceptions. (Extract of *Hunterian Lectures*.)
7. Power. *Veterinarian*, 1897, LXX., p. 648.
Extract from *Archiv. f. Wissensch. und prakt. Thierheilkunde* of paper by Rackow of Berlin.
Horse, nine years of age, colic, passed per rectum inverted cæcum on the tenth day, recovery. Variety, colic.
8. Dorn. *Veterinarian*, 1901, LXXIV., p. 548.
Ox, 10 inches invaginated. ? small gut. Extract from the *Wochenschrift für Thierheilkunde*, etc. Operation, reduction, recovery.
9. Allen. *Veterinarian*, 1901, LXXIV. p. 71.
Dog, four months old, death three to four days. *Post-mortem*—Jejunal intussusception 6 inches long. Variety, enteric.
10. Macqueen. *Veterinary Journal*, 1892, XXXVI., p. 114. Also in *Veterinarian* of same year. Says that they usually consist of small gut invaginated into small gut, *i.e.* enteric. (The reported cases confirm this, though not so simply as would appear in dogs.)
11. "Hirudo." *Veterinary Journal*, 1895, XLI., p. 36.
Mare, eight years old, death, cæcum into colon. Variety, colic.
12. *Veterinary Journal*, 1902, LV., N. S. VI., p. 133. Chiefly affects cæcum and colon. (Apparently only true of older horses.)
13. Webster. *Journal of Comparative Medicine*, 1896, XVII., p. 612.
Gelding; pain, abdominal distension, etc. Death in forty-eight hours. *Post-mortem*—Three feet of ileum invaginated into itself and then through the ileocæcal valve. Double intussusception, ileocolic-colic.
14. Michener. *Journal of Comparative Medicine*, 1897, XVIII., p. 779.
Colt, three months old, colic etc. Operation, and 2 feet 3 inches of small intestine reduced from invagination, recovery. Variety, enteric.
15. Lyman. *Journal of Comparative Medicine*, 1896, XVII., p. 550.
Colt, not three weeks old. ? of small gut, gangrenous, death. Variety, ? enteric. Collection of fluid between the layers of invagination.
16. Butterfield. *Journal of Comparative Medicine*, 1893, XIV.
Devon ox, small gut apparently, easily reduced, recovery. Variety, ? enteric.
17. *Archiv. für Thierheilkunde*, 1898, XXIII., p. 336-339.

Horse, nine years old, colic, passed cæcum invaginated in the cæcum. recovery, variety colic.

18. *Receuil de Méd. Vétérinaire*, Tome 7, 8 ser., 1900.

Long paper dealing with signs, symptoms, treatment, etc. Mentions five or six cases in dogs.

19. Petit. *Receuil de Méd. Vétérinaire*, Tome 8, 8 ser., p. 191, 1900.

Several invaginations of small gut into the colon. Says that it is quite a common lesion in dogs. Seven observations recently found. *Post-mortem*. (? Intussusceptions of the dying). Cases show 10-20 cm. of small gut into the colon. Variety ileocolic.

Extract from *Bull. de Soc. Anat.*, Paris, 16th Nov. 1900.

20. Petit. *Receuil de Méd. Vétérinaire*, Tome 2, 8 ser., 1895, p. 569. *Extract of Veterinary Magazine*, 1895.

Dog, small gut into small gut, then prolapsed into the large, and finally second colic intussusception super-imposed. Double ileocolic variety.

21. Mauri. *Receuil de Méd. Vétérinaire*, Tome 1, 8 ser., 1894, p. 299. *Revue Vétérinaire*, July.

Horse, three years, small gut into cæcum, variety ileocolic. Quotes as recording cases, Bouley Jeune, Reyual, and Mitant. Variety ileocolic.

22. Hobday. Unpublished.

From litter of chow-chow puppies, four fatal cases of enteric intussusceptions.

The results of this series may be summed up as follows:—

(1) Three recorded cases in cattle, nine in horses, eighteen in dogs.
(2) Of these three are recorded as double, and eight or nine might very well have been so.

(3) Records of over thirty cases, many of which cannot be consulted in the original, and many are scanty in anatomical details.

(4) Seven cases were purely enteric—two in colts, and five in dogs. Ten cases were ileocolic—three in horses, seven in dogs. Five cases were purely colic, all in horses.

(5) In seventeen cases the invagination arose in the small gut. In five cases the invagination arose in the large gut. And in no case did it begin at the ileocæcal valve.

(6) In dogs the intussusception always begins in the small gut. In horses, when very young, it starts in the small gut; when older, in the cæcum. This is no invariable rule, and may be only apparently true.

It is thus seen that the veterinary observations entirely confirm the suggestion that I made for human intussusceptions (St. Thomas' Hospital Reports, 1901), that they do not begin at the ileocæcal valve, *i.e.*, the ileocæcal variety, but in the small bowel above it. This small bowel becomes prolapsed through the ileocæcal valve, and finally engages in it, and, becoming tightly grasped, a second colic intussusception may be superimposed on the original ileocolic. Hence the commonest double intussusception in man and animals is the ileocolic-colic. But should the enteric intussusception begin far above the valve, its intussusceptum may become too œdematous to get through the valve, which will be pushed forward by it. The resulting double intussusception will be enteric-ileocæcal.

The following tables may be arranged, the varieties being in order of the probable frequency.

Single Intussusceptions.

<i>Man.</i>		<i>Definition.</i>
Colic		Large gut into large gut.
Ileocolic		Small gut through the ileocæcal valve.
Enteric		Small gut into small gut.
Ileocæcal		Origin at valve, which forms apex of intussusception.
<i>Animals.</i>		
Ileocolic		As above.
Enteric		As above, especially in dogs.
Colic		As above.
Ileocæcal		Undescribed.

Double Intussusceptions.

<i>Man.</i>		<i>Definition.</i>
Ileocolic-colic		The most common of all. Ileocolic as above, with secondary colic intussusception.
Enteric-ileocæcal		Next common.
Rest uncommon except the colic-ileocæcal form.		
<i>Animals.</i>		
Ileocolic-colic		The only form described.

One of the chief distinctions between man and animals lies in the apparent proneness of ileocolic intussusceptions to take upon themselves a secondary colic invagination, whilst in animals this is not so marked. The result is that the double ileocolic-colic intussusception is most probably the commonest in man, whilst the single ileocolic seems the most common in animals. From examination of the recorded cases, it is impossible to say whether this distinction is real or only apparent.

A small point may also be noted, that in animals, possibly on account of the larger size of the parts, *e.g.*, horses, noticeable collections of fluid are found between the peritoneal layers of the intussusceptum or invaginated gut.

In man there exists an uncommon but not unknown variety of double intussusception, which begins by the invagination of the cæcum, and so blocks the ileocæcal valve, usually incompletely, and causes a secondary ileocæcal invagination on account of the peristalsis of the small intestine. This is called colic-ileocæcal. Two or three cases in veterinary literature may have been of this kind, but the descriptions are not full enough in detail to warrant a diagnosis being made.



EDITORIAL ARTICLE.

THE SHORTHORN SOCIETY'S MALEDICTION OF
TUBERCULIN.

IN reading the report of the recent meeting of the Shorthorn Society, admirers of the works of Thomas Ingoldsby cannot fail to be reminded of the "Jackdaw of Rheims." Apparently two lines from that immortal poem, with one or two words altered, might serve to describe the most important transaction at the meeting in question :—

"The breeder rose with a dignified look,
He called for his candle, his bell, and his book.
In holy anger and pious spleen
He solemnly cursed tuberculin."

The crime with which the object of this malediction is charged is that it has yielded results misleading and unsatisfactory to the breeders of pedigree shorthorns, and the Shorthorn Society has solemnly pledged itself "to resist the harassing, expensive, and unnecessary conditions imposed by certain foreign and colonial Governments, which greatly restrict the trade in pedigree cattle." Needless to say, the condition which, in the opinion of the members of the Shorthorn Society, deserves the strong epithets introduced into this resolution, is that animals intended for importation into foreign countries must be free from tuberculosis, as indicated by the tuberculin test. One naturally wonders whether, in banning this condition, shorthorn breeders ground the denunciation mainly on the unreliability of the test for the object which importing countries have in view, or on the unwisdom of the Governments of those countries in availing themselves of the best means at present known of ascertaining whether an animal is the subject of tuberculosis or not.

If the members of the Shorthorn Society have really succeeded in persuading themselves that the tuberculin test is so unreliable that it ought to be abandoned—and upon the whole it appears to be probable that that is the opinion of the majority of them—one can only regret that they have arrived at a conclusion so absolutely opposed to the results of an experience which is already immense. No doubt it is for the moment a comfortable frame of mind for the breeder who has had the misfortune to have some of his apparently healthy animals condemned under the test when non-reaction to tuberculin is made one of the conditions of the sale ; but no one who

has made an unbiassed examination of the available evidence with regard to the general reliability of the test can doubt that it would be wiser for the owner in such circumstances to accept the verdict of tuberculin, and recognise that in all human probability his reacting animals are really tuberculous. Furthermore, we hope we may, without giving offence to the members of the Shorthorn Society, say that their own convictions on this matter, however loudly asseverated, will avail nothing to break down the opinion now held by all whose personal interests do not blind them to the truth—that the tuberculin test, when properly carried out, is one of quite remarkable accuracy.

Perhaps, however, the Shorthorn Society do not so much impugn the general reliability of the tuberculin test as the policy of importing countries in attaching any importance to freedom from tuberculosis on the part of imported animals, and in refusing to accept every outwardly healthy animal as absolutely free from that disease. If that is the ground which they take up, one can only say that, while the immediate advantage which would accrue to the owners of tuberculous herds if the tuberculin test were abandoned is perfectly obvious, we have nowhere yet seen set forth the benefits which an unrestricted trade in tuberculous pedigree cattle would confer on the importing countries.

It is surely the merest foolishness on the part of breeders in this country to ignore the fact that the countries which insist upon the application of the tuberculin test to all cattle imported for breeding purposes thereby succeed in excluding many tuberculous animals that might otherwise gain admission and introduce the disease into herds previously free from it. In saying this we do not forget that on the tuberculosis question importing countries have also two hostile camps. That, however, is only what might have been expected, for in both exporting and importing countries the measures enforced in the general interests of cattle owners may seriously interfere with the business operations of a minority. Here, as in many other cases, ignorance and the bias of self-interest account for the diversity of opinion. We think, however, that British breeders of pedigree cattle would do well to recognise that there is not the most remote probability that any single Government, either foreign or colonial, will ever be induced to admit cattle from a country in which tuberculosis is common without insisting upon the application of the tuberculin test.

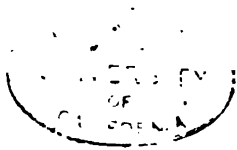
In saying this we are reminded of the fact that at least some countries which insist upon the tuberculin test being applied to imported cattle make an exception in favour of animals from the Channel Islands. The fact is one over which the members of the Shorthorn Society might be advised to ponder. In the first place, they would do well to reflect why this concession was made to breeders in the Channel Islands. It is well known that it was from no motives

of generosity towards the breeders in these islands, nor was it granted because of any pressure which these breeders brought to bear on the Governments concerned. The exception in favour of Channel Island cattle was made simply because the information in the possession of the importing Governments convinced them that the cattle in question were free from tuberculosis, and that in their case the tuberculin test was therefore unnecessary.

But it is still more interesting to note that it was the uniform failure to obtain reactions to tuberculin in Jersey and Guernsey which first brought to public knowledge the fact that among the cattle bred there tuberculosis is a practically unknown disease. We have ourselves had brought to our notice the results of tuberculin testing applied to hundreds of Channel Island native cattle, drawn from many different farms, and we have never yet known a case in which a decided reaction was obtained. It is known, however, that the opinion that these cattle are practically free from tuberculosis does not repose solely on the results obtained by testing large numbers of them with tuberculin. It is confirmed by the observations made by veterinary surgeons on the native cattle which die on the islands or are killed there for food.

As we have already said, these are facts which British breeders of pedigree stock might with much advantage to themselves take into serious consideration. In the first place, let them endeavour to find an honest answer to the question why Jersey and Guernsey cattle remain free from tuberculosis as long as they are kept on their native islands. Would the members of the Shorthorn Society consider it safe or prudent on the part of the Channel Island authorities to admit cattle from other countries without demanding any guarantee of freedom from tuberculosis save that they are in good condition and display no outward sign of being infected?

In the second place,—and this question they will probably find even less palatable than the first,—if, as there seems to be good reason to believe, Channel Island cattle are at present free from tuberculosis, how do they reconcile the non-reaction of these animals to tuberculin with their cherished belief that the tuberculin test is entirely untrustworthy? We venture to affirm that the two things are irreconcilable, and that the failure of Channel Island cattle to react to tuberculin is evidence so strong as to be almost conclusive that the tuberculin test is, as we have previously said, one of quite remarkable accuracy.



Reviews.

Two Years with the Remount Commission. By Harold Sessions, F.R.C.V.S., etc. London: Chapman and Hall, Ltd., 1903.

THE author of this book deserves to be complimented on the manner in which he has carried out his task. Having served for two years and a half on three Remount Commissions, and in that capacity visited Spain, the Argentina, and the United States, he is obviously entitled to speak with considerable authority on all matters relating to the providing of horses and mules for war purposes.

After describing the constitution of the Remount Department of the British army, the author devotes a chapter to the horse supply of the world, and then proceeds to consider in more or less detail the source from which the army drew the supply of remounts during the late Boer war, and the circumstances connected with their purchase, shipment, and transport to South Africa. In all these matters the author has much to say that is both interesting and instructive. The tale which he tells is not very flattering to the Government or the War Office, and the latter department of State comes in for a good many hard knocks. Mr Sessions, however, is a less savage critic of War Office methods than some others who have written on the same subject, and he is fair enough to recognise that many of the difficulties with which the authorities were confronted during the late war were almost unprecedented in character and in magnitude. If the facts he mentions excites one's wonder at the blunders that were made, they also give one the impression that on the whole the various Remount Commissions carried out the work entrusted to them in a way that reflected credit on the officers concerned. A great deal has recently been heard of the extravagant prices paid for remounts, but it may be noted that Mr Sessions' inquiries showed that the German and American Governments when purchasing horses in the United States during the China war had paid as much or more than was paid for British remounts in the spring of 1902. In discussions regarding the remount positions the "middleman" has come in for almost universal denunciation, but Mr Sessions' opinion appears to be that direct purchase of large numbers of horses for war purposes is generally impracticable, and that the middleman must be regarded as a necessary evil.

We need hardly say that the book has a special interest for veterinary surgeons, but even the layman who is neither horseman nor soldier will find in it much that is of interest to him. The book contains a large number of illustrations, mostly reproduced from photographs taken by the author.

The Veterinary Pharmacopœia and Manual of Comparative Therapy. By G. Gresswell, M.A., and C. Gresswell, M.R.C.V.S. Second Edition. London: Baillière, Tindall & Cox. 1903.

For the purposes of a review this book is best considered from two different standpoints, namely as a pharmacopœia, and as a guide to veterinary therapeutics.

As a pharmacopœia it is good in many respects. Now-a-days it is often useful to have the English (B.P.) and the American (U.S.P.) methods and preparations side by side, and this the book gives. The authors also give a number of very useful non-official formulæ of preparations adapted to

veterinary practice, many of which are new to us, and some appear distinctly good. Generally this part of the book is accurate, although a serious error occurs with regard to Fleming's tincture of aconite. This preparation is stated to be four times as strong as the B.P. official tincture, whereas, since the B.P. preparation is made with 1 of aconite root to 20 of rectified spirit, and Fleming's tincture with 1 of the root to $1\frac{1}{2}$ of spirit, the latter preparation is over thirteen times as strong as the official one. It is unfortunate that it has not been seen fit to include many of the more recently introduced agents, and, in fact, some quite old ones too. Among other omissions we note, barium chloride, chinisol, creolin, lysol, caffeine, and arecoline.

Turning to the therapeutics part of the book, we regret that we are unable to recommend it. The subject is often dealt with in a scrappy fashion, the explanations are often brief at the expense of being lucid, frequently no explanation of the actions is given, and in several cases nothing at all is said with regard to the actions and uses of the drug. This tendency to omit the *why* and the *how* of the actions of an agent is far too common among students of materia medica to be encouraged by a text-book; for, if allowed to develop, the result is pure empiricism. In other places the actions described are largely those produced in the human subject, and are of little or no interest to the veterinary reader (*vide* potass brom.).

Some of the pathological allusions are curious, as, for example, "scarlet fever" of the horse, whatever that may be, and also the statement that malignant pustule (anthrax) and texas fever are due to the same virus. We should imagine, too, that a good deal of sodium sulphite would have to be given to prevent anthrax if an animal were to be submitted to infection.

On the subject of anæsthesia we are still told that the dog is a bad subject for chloroform, whereas the contrary has surely been proved up to the hilt by Hobday. Again, in the case of opium the general statement is made that this drug causes contraction of the pupil of the eye, whereas its action varies considerably in the various animals, both the horse and cat showing dilatation of the pupil. Then too, as antiseptics, we certainly doubt the *efficiency* of both boric acid and borax, and we see no reason why sodium sulphocarbolate (a very mild astringent and slight antiseptic) should be preferred to carbolic acid in cases of flatulence or tympanites.

In conclusion, we are bound to say the whole treatment of the subject of therapeutics is disappointing, both on account of inaccuracies, and, especially, because of want of clearness and explanation.

The Castration of Cryptorchid Horses and the Ovariectomy of Troublesome Mares. By Fred. T. G. Hobday, F.R.C.V.S. Edinburgh & London: W. & A. K. Johnston, Ltd. 1903.

To the readers of this Journal Mr Hobday is well-known as one of the most indefatigable workers in the field of veterinary surgery. In connection with the two subjects dealt with in this monograph he has had an amount of experience that enables him to speak with authority, and he has done good service in placing the fruits of that experience before the profession. The book extends to a little over 100 pages, and it contains thirty-four illustrations. Hitherto both the operations dealt with have been generally regarded as too difficult to be undertaken except by a "specialist," and that may continue to be the case in the future also. We see no reason, however, why any competent veterinary surgeon who has carefully studied Mr Hobday's directions need hesitate to undertake either operation, and we may add that no one who contemplates undertaking either operation can afford to do without this work.

Annual Report of Proceedings under the Diseases of Animals Acts, etc., for the Year 1902.

THE Annual Report of the Board of Agriculture for the past year opens with a paper from the pen of the Chief Veterinary Officer on Contagia, in which a brief account is given of the discoveries that have during the last half century given precision to our notions regarding the actual cause of the contagious diseases of the domesticated animals. This is followed by an account of the incidence of the scheduled contagious diseases during 1902, this being also contributed by the Chief Veterinary Officer. The mysterious outbreak of foot-and-mouth disease which occurred in Kent in the month of March is described at length, and it is admitted that the most searching inquiries failed to give the clue to the introduction of the disease. In the paragraphs relating to swine fever, complaint is made that there is often considerable delay in the notification of outbreaks, and that in many instances pigs are ill for days, some of them even dead, before the facts are brought to the knowledge of the Board. We have no doubt that this complaint is well founded, but pig-owners have also been known to complain that after intimation of the supposed existence of swine fever has been promptly given the measures appropriate to the suppression of the outbreak have not always been carried out by the Board with all the celerity that is desirable.

The Assistant Secretary also contributes an account of the work of the Animals Division for the year, and the Report concludes with the usual Statistical Tables.

Department of Agriculture and Technical Instruction for Ireland. Report of Proceedings under the Diseases of Animals Acts, for the year 1902.

THE report by the Chief Inspector of the Veterinary Branch of the Department shows that during the past year the efforts made to exterminate swine fever have had a gratifying measure of success. During the twelve months the outbreaks detected numbered 166, being a decrease of 54 outbreaks as compared with 1901. The total outbreaks of glanders in 1902 numbered ten (as against five in the preceding year) and most of these are laid to the charge of diseased animals imported from England and Scotland. In one of the most serious outbreaks mallein was usefully employed to ascertain how many of the in-contact horses were infected, and it was found that eventually the animals which responded to the first test ceased to react.

CLINICAL ARTICLES.

CANCER CASES.

By C. CUNNINGHAM, M.R.C.V.S., Slateford, Midlothian.

IN these days of "Cancer Research" and keen investigation the veterinary surgeon should help. Possibly he cannot do much. Carcinoma does not play such a big part among the diseases of animals as in those of man. Still, hidden away in casebooks or borne in mind are doubtless details of many interesting cases, and these with the occasional living subject may be of some little service.

The terrible malignity of the disease in human beings, every one knows. Brilliantly successful operations for cancer are, of course, often performed, but they frequently fail.

Such operations are almost the every-day work of high-class surgeons, done frequently too in hospital without fee or reward—all honour to them. Equally difficult and much more dangerous operations are frequently performed by members of the veterinary profession; but, as every one knows, for work of this kind, as a general rule, they have neither the opportunity nor the suitable subject. In private practice, again, the fee of any surgeon of note for a big operation ranges from ten to fifty guineas, in higher circles £100 to £1000, and, where "Crowned Heads" are concerned, double and treble the latter amount. After all is said and done, "a man is a man and a beast is a beast," and will be to the end of time. The so-called equality of the two professions is surely in many of its aspects foolishness.

Of the extremely malignant type of cancer in the lower animals, I have only met with two plain and well authenticated cases,—one in the udder of a mare, in which the disease, slow and mild at first, increased and spread alarmingly at the finish, destroying life. The organ when roughly put on the scales weighed 30 lbs. The second case involved the cheek, among the tissues and bones on the side of the face, of a shorthorn cow, destroying the eyeball, eating into the bones forming the orbital cavity, and spreading more or less on the superior and inferior maxillary bones; a disgusting foul-smelling object, which, if it had been allowed to live long enough, would have shown as fell and dire cancerous results and aspects as any met with in the human subject. Sections from the diseased growth in the mare's udder were very kindly examined by Principal M'Fadyean, and pronounced "Carcinoma—the only case of real carcinoma in the udder of the mare he had examined"—up to that date, January 1895. I was also favoured by an opinion from the Pathological Department, University of Edinburgh, and by that of a medical friend; "Carcinomatous, a malignant growth very liable to recur": "Cancer, no doubt about it." Sections too from the diseased mass on the cow's face were also kindly examined by Professors M'Fadyean and Stockman, and certified, not as might have been supposed actinomycosis, but true carcinoma.

Details of the cases, are as under:—

CASE I.—Mare. Summer of 1894. Brown Clydesdale, sixteen hands, eleven years, been used at farm work, and having a pedigree as a brood mare. Has had five foals, is now in full milk with foal at foot, in good condition and health to appearance; but while the near quarter of the udder is sound and right, yielding an abundant supply of good rich milk, the off quarter is a mass of disease, presenting an appearance new to me in the udder of a mare, but approaching somewhat that occasionally seen in long standing cases of scirrhus and fistulæ of the spermatic cord in geldings. A hard dense tumour-like mass clinging firmly to abdomen and thigh; "pitted" and "dimpled" all over with about a dozen small depressions. In some of these depressions were openings of slender sinuses leading into the substance of the gland, and from these orifices a discharge of thick viscid gluey fluid, which, drying on the surface of the skin and smeared over the nose and lips of the foal, presented a very nasty appearance.

The owner expressed himself from the first and all through as opposed to surgical operation. During the nursing of the two preceding foals the disorder in the udder had been observed to a slight extent; as the primary cause, something was hinted about an injury from barbed wire, but nothing certain known. As a slight experiment in treatment, full doses of iodide of potassium (and of iodine occasionally) were given morning and evening, and an attempt made to syringe sinuses with tinct. iod., once a day, but without any apparent effect, good or bad.

At the end of the grazing season, the foal was found to be a strong, healthy subject, but the mother had made rapid strides in the downward grade—thin and low in condition, mass in off groin very much larger, substance of near quarter invaded. Everything pointed to a fatal termination before many months or weeks. The owner still would not entertain the idea of operation. "His mare would die in our hands, he would have no cutting but would work the mare quietly for a time, and destroy her." In a short time softening and sloughing between the teats set in, causing a large open ulcerated sore, almost black in colour, with everted edges, and peculiar foul-smelling discharge. Occasional hæmorrhages followed, once to an alarming extent, any dressing or interference only making matters worse; and, when scarcely able to walk, she was destroyed early in January 1895.

Section through the diseased mass showed that the glandular structure of the quarters had almost disappeared, what little remained serving only as a matrix or bed for the growth of a hard firm greyish-white, almost glistening and mother-of-pearl substance, "cutting like cartilage," or "a hard potato"; and the difference of the internal structure and the external black ulcerating sore with its disgusting discharge was very striking. On examining the mass to see if surgical removal would have been possible or feasible during life, the vessels of supply and exit at the upper and central parts of the glands were certainly of a size to make one ponder and pause; and, to increase the difficulty, certain very large blood vessels were found coursing where one would scarcely expect to find them. Still, with a liberal supply of knives (sharpened to finest edge—the structure was tough and not easily cut into), artery forceps, ligatures, and with sufficient skilled assistance, the operation would certainly have been a formidable one, but might have been successfully carried out in this, as in other recorded cases. It could scarcely have been much more than the removal of a large scirrhus mass from a carriage gelding's groin, which two of us once attempted, when three or four arteries of the size of goose-quills, very close together in the very centre of the hard unyielding mass, sent their streams into the air six or eight feet in a circle, spraying the man with the chloroform bag at the horse's head. There was no time or chance for ligaturing in that case. Compression with the hand, big pledgets of wool and fine tow, and deep and firm stitching of the wound controlled the hæmorrhage, and the horse did fairly well for a couple of years. Recurrence, however, after that time set in to such an extent that the horse was shot. Section through that big tumour showed no ulcerating surface, no dull "mother-of-pearl" cartilage aspect, but a yellowish fibro-cartilaginous appearance, and, singularly enough, few if

any suppurating centres. It was not, I think, the seat of true carcinoma, though in its last stages in malignancy it seemed allied to it.

I omitted to mention that at the *post-mortem* examination of the mare we found on opening the chest that degenerative change had set in on the lower border of the off lung, and the parts in contact—soft and dark in colour, and easily broken up; a fresh centre of disease.

I may also state that three of this mare's progeny are still on the farm. Two of these mares have been bred from, but none of the stock so far as I am aware have shown any unhealthy or cancerous tendency.

CASE II.—Cow. September 1899. Shorthorn, white, six years or so, bought four months previously. Small warty excrescence observed then on off cheek, but looked on as a wart, and little attention paid to it. Cow tied up to stake and wall of cow-house, to the off side, and warty excrescence "out of sight, out of mind" to a careless owner, till now there exists a large circular flattened cauliflower-looking substance or tumour, seven inches in diameter, and about two in thickness, black and ulcerated, saucer-shaped, with rounded everted edges, growing from the tissues of the cheek and side of the face, with pedicles hanging from its lower part. The stench of the discharge could be smelt from a considerable distance. (Singularly enough, by the way, the County Sanitary Inspector had paid his official visit to the dairy a few days before, and detected nothing amiss. The smell might have told him, though standing next the wall the diseased mass was out of sight. Good worthy tradesman or railway servant as he used to be, it did not appeal to him as his duty to submit each of the dozen or so cows in that dairy to a careful examination. Another instance of the beauty and benefit of layman-inspection.)

Examination of the bones of the head during life and after death (see fig.) showed that surgical interference was worse than useless, and recovery impossible. Section of the diseased mass showed to the naked eye an exactly similar structure as in the mare's udder. In one of the greatly enlarged glands of the throat, too, three separate centres of growth, each of considerable size, showed their white pearl structure very distinctly in the pale reddish glandular tissue in which they were embedded.

Another instance of real carcinoma, though not of great malignancy, I was favoured seeing in the practice of Mr Aitken, Dalkeith. White cob, aged, fat, and comfortable—evidently had pleasant times during life—had for a considerable time showed a persistent discharge from near nostril, for which he had been treated without benefit. In the end he was trephined, but on the circular bit of bone being removed no pus could be seen, and very little of the sinus itself, its cavity being apparently occupied by a smooth gristly sort of growth, the apex or cone of which could be easily felt and broken down by the finger inserted through the circular aperture. A small portion of the growth was forwarded to Principal M'Fadyean and pronounced carcinomatous. On the slaughter of the animal the cancerous growth on a transverse section of the head could be seen growing from the floor of the sinus and almost obliterating its cavity.

The nasty repulsive bad smelling growths or excrescences not

infrequently, I believe, met with on the eyelids, generally the lower, of cattle and dairy cows, one would almost think partake somewhat of a cancerous tendency. One such which I removed with the ecraseur some time ago, in its black ugly look and general appearance, blocking up the eye and hiding it completely from sight, resembled very much the true cancerous tumour on the cheek of the shorthorn cow already mentioned. The discharge and peculiar smell too seemed much alike. The case in the hands of a would-be-economical farmer had gone on for months, and I very much feared I had another case of cancer to deal with. Recovery after operation, however, was so rapid, complete, and lasting, as to negative the view.

Another instance of recovery from what we deemed, and in fact from what was pronounced, carcinoma, occurred last year, 1902, in the



Skull of cow, showing effects of carcinoma.

case of a valuable farm horse, the seat of the disease being the lower eyelid, and to a certain extent the nictitans and cornea. Big upstanding horse, over seventeen hands, about ten years old, cost at five years £100. Had the misfortune two or three years before to have the eyelashes and part of the lower lid torn off, and the eye was ever afterwards troublesome; swollen and tender, with yellowish thick discharge, bleeding occasionally, etc.

On examining him for the first time in the warm weather of last summer, I found the lower lid very much swollen and indurated, projecting considerably, membrana nictitans in much the same condition, half of it having apparently been torn off at the time of the injury, cornea clouded, conjunctiva very much thickened and congested, with a small circular patch of fungous growth at junction of cornea and sclerotic on the lower side.

Treatment seemed to have no effect whatever on the case, though in darkened loosebox here for three weeks. Fearing malignancy, I applied cocaine and cut two or three tiny sections from the inside of the lower lid, and under the microscope carcinoma was diagnosed. Thinking the case incurable, I had what proved to be a very useful and effective "blind" to protect from sun and dust sewed on to his bridle, and gave the man some strong astringent lotion and a little Jeyes' Fluid, with a little *douceur* to keep up his courage against the idea of cancer infection, and to enlist his better care and attention; and, to my great though pleased surprise, under this simple *régime*, as the cold weather set in, the eye began to improve, and now has completely recovered, though only a vestige of the lower lid remains, the result of the primary injury.

One other extremely strange case of very long ago I remember, in which the idea of sarcoma or carcinoma obtruded, but was not tolerated by the microscope. An enormous mass of 238 lbs., or 17 stones, of diseased growth found at the *post-mortem* of a two-and-a-half years old Clydesdale colt, involving liver, spleen, and mesentery.

Bought at a November Fair, the colt soon after fell into low health and condition. Further than the presence of some dropsical fluid in abdomen, possibly also in chest, and certainly in pericardium, I could not get. Two or three veterinary surgeons and one Professor called in consultation, were in like case. No colicky pains nor anything to indicate the presence of such an enormous mass in the abdomen.

Under the microscope in the late Principal William's hands, in those early days, it was declared a simple fatty tumour. In outward appearance it looked more like cancer or some malignant growth.

Of the other forms of carcinoma, medullary, colloid, etc., I do not remember any well-marked instances.

Tubercular tumours from the horse I have seen on section simulate very closely those of carcinoma. Almost the same dull glistening white or pearly grey appearance, though those of tubercle are considerably softer and more easily cut into.

In a very marked case of tuberculosis in a middle-aged farm horse, allowed to live and run its full course, characterised too by the stiff neck and arched back and skeleton appearance of such cases, the abdomen on being cut into was quite a picture. Omentum and mesentery studded with thousands, if not millions, of small tubercles, singly and in little masses, presenting when spread out quite a web or lace-like appearance. The spleen showed as a great big whitish-green "marled" mass of 19 lbs., blocked almost to rupture with tubercular masses of all sizes. The diaphragm was of the same weight, and also loaded with deposit. Not much wrong with the lungs, only a little fringed round the lower edges with small pedicles dependent. I had the curiosity to cut into and divide hundreds of these tubercles, big and small, and in none of them did I find a softening centre, or any trace of caseation or of calcareous deposit, but only the pearly structure not unlike that of cancer. Tubercle in the horse resembles somewhat in this respect what I have met with in outbreaks of tuberculosis in pigs.

Cancer, I note from the public papers, is said to be much on the increase, 3000 deaths recorded in Ireland last year, and England and Scotland in like ratio. One alarmist Professor a year ago pro-

phesied that in ten years there would be more fatalities from carcinoma than from consumption, small-pox, and typhoid fever combined. This in human beings! How stands the matter among the domesticated animals? A worthy subject of inquiry surely.

CASES OF ANTHRAX WITHOUT MARKED ELEVATION OF TEMPERATURE.

By G. H. GIBBINGS, F.R.C.V.S., Tavistock.

I REGARD the veterinary surgeon who is called to a case of anthrax prior to death as singularly unfortunate, especially if he is a stranger to the district.

The symptoms of the disease are so obscure and variable that even the most experienced and careful practitioner is liable to err.

The most constant indication of anthrax in cattle is the presence of small clots of dark-coloured blood mixed with rather loose fæces, and this is usually associated with a high temperature, but the latter is not essential when the animal is visibly ill. I do not dispute the value of the thermometer in diagnosing the disease during its incubative period; but when the bullock is in such a condition that the owner seeks the services of the veterinary surgeon, then too much reliance should not be attached to what may be termed the negative evidence of the temperature.

A few years ago I was requested to attend a cow. On my arrival at the farm the owner stated she had refused her food in the morning, but now appeared much better and had just devoured a couple of mangolds.

As prior outbreaks of anthrax had occurred on this farm, I was particularly careful in my examination. The fæces were dark in colour, rather fluid, and stained with blood. The cow had a depressed appearance, but to my great astonishment the temperature was only 103° F. After prescribing for the animal, I requested the owner to report to me the following morning. The same evening I received a message stating that the cow died a couple of hours after I left the premises.

I made a *post-mortem*, and found it to be a typical case of anthrax.

In this case I was altogether misled by the thermometer, and had I left this precious instrument at home I should certainly have warned the owner of my suspicions.

The latter end of April I received a hurried message from a poor man stating both of his cows were very ill. I found one dead, and the appearance of the carcase was very suspicious of anthrax. A portion of the ear was forwarded to Prof. M'Fadyean, and the reply came "undoubtedly anthrax."

The other cow located in the same shed refused all food, was scouring profusely, stiff in her gait, and grunted occasionally during movement. Temperature only 102° F. She gradually improved and was convalescent in a week.

I know it may be argued there is no positive proof of the existence of anthrax in this cow, but, considering these were the only two cows

the owner possessed, that they were fed alike, grazed in the same pasture, and were in close contact in the shed, it would be rather a remarkable coincidence if she were the subject of any other disease.

A CASE OF BOTRYOMYCOSIS, OPERATION AND RECOVERY.

By Arthur R. ROUTLEDGE, F.R.C.V.S., London.

Subject.—A brown van mare, about fourteen years old.

History.—For the last three years has had a growth in front of and slightly below the elbow joint, gradually increasing in size. When first observed the tumour was about the size and shape of a tomato, hard, painless, and freely movable, and seemed to invade the skin and subcutaneous tissue only.

The hair over the affected area was scanty, bristly, and erect. The skin studded with small elevations about the size of a bean, which came up as boils, burst, and healed up, leaving a fibrous thickening, or being replaced by other small abscesses.

Occasionally the growth became rapidly larger, and the abscesses were lanced and a biniodide of mercury blister applied, when a slight temporary reduction took place, only to be followed by progressive increase in size.

Eventually the tumour obtained the size of a turnip, and so interfered with the action of the extensors of the forearm as to cause lameness.

Diagnosis.—Botryomycosis.

Operative treatment the only means of relief.

On 1st November 1902 the mare was cast, chloroformed, and the growth removed with antiseptic precautions.

Operation.—An elliptical incision including the affected skin was made, and the tumour dissected from the face of the extensor muscles. During excision two large abscesses in the depths of the growth, each containing about 4 ozs. of pus, were accidentally evacuated.

The edges of the wound were approximated as far as possible by seven tape sutures, which had to be taken out on the seventh day, as they were cutting through the skin and becoming embedded in the muscles, causing excessive granulation.

The wound was dressed with chinosol 1:500, alternated with carbolic acid 1:40, chloride of zinc 1:40, or liq. antim. chlor., as circumstances indicated.

There was considerable discharge, which by 15th November was reduced to a minimum, and the wound from this date made an uninterrupted recovery.

The mare was in slings for about a month, and resumed work 9th February, having been under treatment fourteen weeks.

The tumour, which weighed 3 lbs. 2 ozs., was sent to Prof. M'Fadyean, and he confirmed the diagnosis.

Up to the time of writing (June 1903) the mare has been at regular work, and there has been no evidence of recurrence.

CANINE HYDRONEPHROSIS ; ASSOCIATED WITH CON- GENITAL ABSENCE OF THE OTHER KIDNEY.

By GERALD LEIGHTON, M.D., F.R.S.E. [Interim Professor of
Pathology, Royal (Dick) Veterinary College.]

THE following interesting, and in some respects anomalous case occurred in the Royal (Dick) Veterinary College during last winter session ; and, as the association of conditions which were revealed on the organs coming into my hands for *post-mortem* examination is very unusual, I have thought it desirable to place the case on record.

The clinical history of the case, I have compiled from the notes of the case taken by the late Mr Joseph Warick, M.R.C.V.S., who was then Hospital Surgeon at the College, and whose untimely death towards the close of the session deprived the veterinary profession of what promised to be a most valuable career. Mr Warick handed me the notes of the case after he had been taken ill, and I reproduce them practically in his own words.

Clinical History.—"On 14th November 1902, a sable-coloured collie dog was brought into the hospital, suffering from an attack of intense diarrhoea. The history given by the owner was that this diarrhoea had been continuously present for the three weeks preceding the date mentioned, up to which time the animal had been in perfectly good health.

"On examination it was found that the dog was in a weak anæmic condition, with a ragged coat, and presenting in general a delicate appearance, although quite lively. The pulse and temperature were normal. The conjunctival membrane was of a pale yellow colour. The abdomen was tucked up, and on manipulation no sign of pain was evinced. The diarrhoeic matter voided was thin, watery, and yellowish, having the consistence of gruel.

"The treatment consisted in the administration of an oleaginous purge to remove any fermenting food that might be in the alimentary tract ; followed by tonics and astringents, which seemed to have but little effect in allaying the profuse diarrhoea.

"The same result attended the administration of stronger remedial agents. A second examination was made to reveal, if possible, the cause of the diarrhoea, but nothing was found beyond the symptoms at first noticed. It was thought possible that the dog might be suffering from *tabes mesenterica*. Some large doses of opium were given in order that some mucus might be obtained for microscopical examination. This examination failed to reveal the presence of the tubercle bacillus. Perseverance in the tonics and astringents had at last the desired effect, and on 11th December 1902 he was discharged from the hospital, apparently quite well and healthy.

"The dog remained well until 20th February 1903, rather more than two months later, when he was again admitted, presenting the same symptoms as before, except that the back was slightly arched, and the movement of walking seemed to be attended with pain. As the animal did not improve under further treatment, it was decided after consultation with the owner, to have it destroyed, and accordingly this was done on 2nd. March 1903."

The above are the notes as given to me, and it will be noted that it

was never possible during life to ascertain the cause of the persistent diarrhoea, nor could any definite diagnosis be made. The *post-mortem*, however, revealed an interesting pathological condition, even if it hardly explained the symptoms which had been present.

Post-mortem.—As was inevitable after such long-standing and persistent diarrhoea, the body was emaciated, but, apart from this general emaciation, absolutely nothing abnormal could be detected until the urino-genital system was examined. The left kidney was entirely absent, and as is usual in these cases of congenital absence of one kidney, the other one, the right in this case, was considerably enlarged. The naked eye appearance, both external and on section, of



FIG. 1. External surface of right kidney.



FIG. 2. Section of right kidney.

the right kidney is so well depicted in the illustrations appended, that a detailed description is unnecessary. The one ureter was of great size, readily admitting the middle finger into its lumen, and it opened into the dilatation in the kidney, which was full of fluid. It was, in fact, a typical hydronephrotic kidney. The papillæ were flattened almost beyond recognition, and, as the result of the progressive destruction of the tissue of the pelvis, a large fibrous sac remained continuous with the lumen of the ureter. At one spot, clearly seen in Fig. 2, on the fibrous wall of the commencement of the dilated ureter there was a disseminated gritty deposit extending over an area of half an inch

square. The single ureter was greatly enlarged throughout its whole length, and the opening into the bladder was of corresponding size. The bladder itself exhibited but little change from the normal, being but slightly distended, the coats neither hypertrophied nor atrophied.

The next step was to endeavour to find the cause of the hydronephrosis. As only one kidney was present, it was not possible to say whether the cause was one which was producing an *unilateral* hydronephrosis, or whether it would have had a similar effect on the other kidney had that organ been present. Proceeding, therefore, by a process of exclusion, nothing was found in connection with the ureter to account for the condition. There was no sign of calculus, contraction after an injury, stricture of any kind, or pressure of any growth upon the ureter. There was no twisting. It was therefore obvious that whatever was causing the backward pressure of the urine and obstructing its out-flow must be either in connection with the bladder or the urethra. No obstruction of any kind was found in the bladder. The urethra only remained. This in itself was normal, but it was found that the prostate was considerably enlarged. Nothing more was found to account in any way for the condition, which must therefore be ascribed to the prostatic enlargement pressing upon the urethra.

Remarks.—The case was obviously one of *acquired* hydronephrosis, the dog being, I was informed, eight years old. The single right kidney had apparently performed the function of both without any obvious difficulty for a long time. The prostatic enlargement was probably of recent development, indeed must have been so. The congenital absence of the left kidney reminds one that in a considerable proportion of all cases of hydronephrosis there is some associated congenital deformity, either in the kidney or elsewhere in the body. The difficulty of diagnosis is great unless the kidney forms a palpable tumour, and, as a rule, pain is not conspicuous except in very acute cases. Moreover, hæmaturia is by no means common, the urine more frequently being normal in amount and constituents. In this particular case one cannot easily see that the *post-mortem* threw much light on the clinical history of the dog, and one is rather driven to conclude that the actual cause of the diarrhoea escaped observation. I had the kidney photographed as soon as possible by Mr Swan Watson, of Edinburgh.

AN ACUTE CASE OF ROARING.

By WILLIAM ASCOTT, M.R.C.V.S., Bideford.

THE following is an account of an acute case of roaring which occurred in a half-bred mare, seven years old. The animal was purchased in June 1902, after I had examined her for soundness, by a client (whom I will call client No. 1) for coaching and general harness work. She did her work very well indeed, and was never sick nor sorry during the five months she was owned by client No. 1. She was shown in harness at our local horse show on August Bank holiday, when the going was very heavy, owing to the previous heavy rains on a newly-made ground; and she won first prize in a class of twelve competitors. She was such an exceedingly nice mare that at the end of the coaching season both client No. 1 and myself

recommended her to a mutual friend (client No. 2), who had also known the mare well for about two years previous to client No. 1 buying her. Although not a huntress, she did a day's hunting on 6th November very well, and on 8th November client No. 2 bought her. I examined her again on that date, and considered her to be sound. I think, therefore, having regard to the history thus far, viz., the examination in June, the horse-show test in August, the day's hunting on 6th November, and the re-examination on 8th November, we may fairly consider the mare was sound in her wind up to this date.

She went on very well at first with client No. 2, but on 28th November the groom called for some powders for her, as he thought she had a cold. A week later he called again to say he couldn't make the mare out at all. Some days she went and seemed to be perfectly well; other days she went without any life, and he almost thought she made a noise like a roarer, but it was only noticeable down hill.

On 10th December she was driven in (2½ miles) for me to test. I drove her, and found she was distressed against hill, and made a noise, but this was much more pronounced down hill and on the level. As she was much upset, I sent her home, and arranged to see her in the stable the next morning. There, at first sight, she appeared to be in perfect health, appetite very good, temperature and respiration normal, but pulse only 32; and when she tried to neigh to another horse we noticed she had lost her voice. She was taken out in the afternoon for a little gentle walking exercise, but when only about a quarter of a mile from home she began to make so much noise that the groom took her back. I saw her again the next morning, and found her just the same as before. On the afternoon of the same day the groom took her out again, but when only about 100 yards away she began quite suddenly to make a noise like a cow (the groom said), and fell down, apparently choking. After a while she got up and staggered back, falling once on the way, and again after reaching the stable.

I was sent for immediately, and found her as follows: Temperature, 101°; pulse, 56; respiration hurried, but no roaring. These symptoms quickly subsided, and on the following three days, i.e., Saturday, Sunday, and Monday, the temperature and respiration were normal, but pulse only 30. The appetite continued to be very good, and she ate all her food on Monday night, but was found dead on Tuesday morning, having apparently reared, fallen backwards, and died without a struggle, the straw being undisturbed.

I made a *post-mortem* examination the same day, and was assisted by Mr James C. Erskine and Mr R. E. L. Penhale. With the exception of the larynx and pharynx (which we sent to Professor M'Fadyean for further examination), and the diaphragm, all the organs, including the brain, appeared to be healthy. The diaphragm was badly ruptured, and the bowels pressing through the rupture into the pleural cavity. This, however, we considered occurred when the mare fell, being of opinion that she reared and fell back during a spasm of the larynx, which, together with the pressure of the bowels through the ruptured diaphragm, caused suffocation.

Professor M'Fadyean reported as follows regarding the larynx and pharynx:—

The mucous membrane of the throat and pharynx was normal except for small hæmorrhages, which probably occurred at the time of death. On the other hand, the left crico-arytenoideus posticus was distinctly paler than the right, although there was no recognisable difference in the bulk of the two muscles. On comparing muscular fibres from the two muscles microscopically, it was found that degenerative changes were already recognisable in the fibres of the left muscle. The fact that the left muscle was not visibly wasted was considered conclusive evidence that the paralysis was of comparatively recent origin; and, although he could not pretend to speak very precisely on the point, he thought it very probable that the condition had not been in existence for more than a month.

The case is obviously of considerable interest, in view of the attempts which have sometimes been made to hold veterinary surgeons legally responsible when horses passed by them as sound have a month or two afterwards been found to be confirmed roarers.

AN OUTBREAK OF HUSK AMONG ADULT CATTLE.

By WALTER WESTERN, M.R.C.V.S., Bracknell.

THE occurrence of "husk," among calves and young cattle is, unfortunately, only too common—so common that the condition is one of little professional interest. In the following instance, however, the age of the animals attacked was so very exceptional as to make it worth while to record the occurrence.

In the autumn of 1901 I was called to see six cows which had within the past few days developed symptoms of illness. On arrival I found that the whole six cows were in-doors, that four of them were very ill, and the other two similarly affected, but to a less alarming degree. All of these animals had a husky cough, which in the worst cow was almost continuous, and accompanied by hurried respiration. The appetite was diminished or suppressed, and the temperature varied from 102° to 105·3°. The symptoms entirely agreed with those usually seen in calves affected with husk, but the rarity of the disease among adult cattle, and the fact that ten heifers and four calves belonging to the same stock appeared quite healthy, made one hesitate to diagnose that disease. However, as it appeared likely that one of the cows would soon die, it was killed and the lungs were forwarded to Professor MacFadyean (who had previously seen the affected animals with me). He reported that the disease was undoubtedly husk, the bronchi containing large numbers of the *strongylus micrurus*. The only lesions present in the lungs were bronchitis, with much frothy exudate in the tubes, and some areas of collapse.

One of the remaining cows died ten days later, but the others gradually recovered.

It was ascertained that up to the time when the cows were attacked they had been at grass during the day. There were four meadows altogether, and both cows and young stock had grazed on each of these during recent months, but never at the same time.

Finally, I may add that the disease is hardly ever seen in this district.

Abstracts and Reports.

E. VON BEHRING'S NEW INVESTIGATIONS ON TUBERCULOSIS.

It has already been announced that Von Behring, when receiving the Nobel prize in Stockholm, in December 1901, stated that he was then endeavouring to find a practical method of protecting cattle against tuberculosis. The various stages which this work has assumed during the last six years are given in a recently-issued pamphlet. On account of the importance of these announcements for veterinary medicine, it appears desirable to shortly indicate their more essential features. As Von Behring states in his introduction, attempts are being made with the assistance of the district veterinary surgeons to discover whether young cattle can be so influenced by a simple and comparatively cheap mode of preparation, that under normal conditions of life they will be protected from the injurious results of tuberculous infection. To secure this protection the following method was tried: '001 gramme of a four to six weeks serum culture was injected into the veins of a five to seven months old calf which had not reacted to tuberculin. Four weeks later the animal was given a dose twenty-five times as great, that is to say, '025 gramme of the same serum culture.

Von Behring declares this method of conferring immunity to be without danger and quite reliable. It is certain that it protects the calf against artificial infection with the bovine tubercle bacillus, but the conditions governing the infection of cattle with tuberculosis in everyday life differ from artificial attempts to convey infection. In the latter case the virus is directly introduced into the blood, subcutaneous tissue, or other artificially opened tissues. Under ordinary conditions of life, however, the bacillus is continually introduced into the digestive and respiratory tracts. Infection with tuberculosis is also favoured by the depressing influences accompanying calving, and by the practice of forcing milk production. Although the above method might, therefore, give perfect security against experimental infection, it might prove entirely inadequate against natural infection in infected byres.

This and other possibilities rendered it necessary that animals immunised in the institute should be exposed to infection under naturally existing conditions. Cows with well-developed tuberculosis (animals with severe cough and pulmonary tuberculosis, tuberculosis of the udder, etc.) were therefore introduced among the immunised animals in Marburg, and in addition immunised cattle were placed on farms where it was known that the danger of infection with tuberculosis was particularly great. Finally, Von Behring utilised the assistance of a number of farmers who offered to have their young cattle protected on the Marburg principle.

The susceptibility of experimental animals for tuberculosis varies greatly. According to Von Behring's extensive records, the most sensitive animals of all are guinea-pigs. Then follow rabbits, dogs, and goats, whilst cattle, at least young cattle (five to eight months old), horses, sheep, and white mice show less susceptibility.

Great variations were noted in the intensity of the toxic powers of the cultures, depending on their origin, treatment during growth, etc.; but only one kind of tuberculous toxin could be discovered. This conclusion, which is founded on numerous and very careful tests of tuberculous toxin, and is supported by the fact that chemical analysis seems strongly to indicate the identity of the chemical toxins found in all preparations of tubercle bacilli, is

regarded by Von Behring as tending to prove the identity of human and bovine tubercle bacilli.

As further arguments, he adduces the absence of morphological and cultural differences of any recognisable value between human and bovine bacilli; the identity of the anatomical-pathological appearances when the two diseases are artificially conveyed to guinea pigs; the possibility of conveying human tuberculosis to cattle, and that of increasing the virulence of human tuberculosis for cattle by suitable cultivations in animals; the fact, established by experiment, that bovine tuberculosis does not necessarily produce "perlsucht" in oxen, and the growing conviction that the lesions identified with the name "perlsucht" are nothing more than a particular result of the chronic course of the disease; the possibility of immunising cattle against bovine tuberculosis by means of human tubercle bacilli and *vice versa*; the possibility of modifying according to the wish of the operator, not only the virulence of a culture from a particular source, but also the macroscopical characteristics of cultures.

As compared with the arguments in favour of the identity of bovine and human tubercle bacilli, those tending towards the opposite conclusion seem steadily to be losing ground.

In order to judge of the occurrence and progress of self-immunisation in the animals treated, Von Behring at first employed injections of tuberculin. He found, however, that under the influence of the artificial (immunising) infection the animal's sensitiveness to tuberculin increased in a manner similar to that of epidemiological infection. As a rule the excessive sensitiveness to tuberculin spontaneously disappeared as soon as the symptoms of infection, viz., fever, loss of weight, coughing, etc., completely abated. Von Behring, however, emphasises the fact that the disappearance of sensitiveness to tuberculin by no means indicates liability to later infection, so that in forming a prognosis the results of the tuberculin test can only be used with very great care. For similar reasons the tuberculin test should only be employed with limitations, because a condition of "tuberculin-immunity" may otherwise be induced, which would make it impossible to rely on the tuberculin reaction as a test for existing tuberculous disease. The tuberculin test must, therefore, not be repeated at short intervals.

As a means of determining whether newly-bought and apparently healthy cattle are or are not free from tuberculosis, the tuberculin test, however, as described by R. Koch, still remains unrivalled. In company with Dr Römer, Von Behring made a long series of experiments with the purpose of discovering a practical "agglutination" test for the early detection of tuberculosis in cattle, but without success.

At the present time Von Behring only utilises human tubercle bacilli for immunising cattle. He employs a standard culture which was obtained in 1895 by infecting a guinea-pig with phthisical sputum, and has since been transmitted many times from guinea-pig to guinea-pig.

Von Behring's description of the staff, buildings, and apparatus for the tuberculosis investigations he is carrying on are very interesting.—(*Deutsche Thiermed. Wochenschrift*, No. 37, 1902.)

NOTES ON THE CAUSES OF PARALYSIS IN THE COW BEFORE AND AFTER PARTURITION.

THE first case described by Ohler was that of a cow which became paralysed after her fourth calf. Calving occurred rapidly, and the after-birth came away on the following day. Peristaltic movements of the bowel could be heard, but

after the animal had been turned on to the other side signs of movement in the rumen were suppressed. On percussion, the animal extended the head horizontally forwards as soon as the instrument approached the neighbourhood of the reticulum. On exercising moderate pressure with the clenched fist at this spot, the animal groaned, and vainly endeavoured to rise. As these symptoms appeared to suggest the existence of a foreign body, Ohler recommended that the animal should at once be slaughtered. This was done. On removing the abdominal contents, he found the usual adhesions which a foreign body sets up, and discovered a broken hair-pin which had thrust its way as far forward as the diaphragm. The sinus it had produced was still lined with granulation tissue, and seemed of quite recent formation, though the reticulum was covered with firm new connective tissue over an area as large as a cheese plate.

As delivery had been easy, the passage of the after-birth had occurred without any complication, and the uterus had contracted. The presence of the foreign body appeared to have been the only cause of this "paralysis."

Ohler noted a similar case in a cow after her third calving. Delivery was very easy, but the after-birth had not been passed on the succeeding day when he was called in. The animal showed symptoms on palpation and percussion of the reticulum almost identical with those just described. As he again feared the presence of a foreign body, the owner proposed to slaughter the animal, but agreed to Ohler's suggestion to wait until the following day. Next day she seemed to have improved, having taken some fluid food, and looking brighter. As the membranes had not then been passed, Ohler removed them manually, and had the vagina and uterus washed out with a disinfectant. The cow appeared to improve from day to day, though she still showed the above-mentioned symptoms on palpation of the reticulum. Rumination was also resumed, but when the animal received hay or other bulky food she groaned, and the disease seemed to become aggravated, for which reason only soft food was given for two or three weeks. After the lapse of this time the cow appeared to have recovered. Fourteen days later, however, she had a mishap, fracturing her ilium, and displacing the stifle joint. She died from internal hæmorrhage. On *post-mortem* examination, Ohler found, in addition to the above-named conditions, the reticulum and part of the liver adherent to the diaphragm. Granulation tissue and fibrous connective tissue united the different parts together. The foreign body (a broken fragment of wire) had almost entirely returned into the reticulum, and it required a comparatively powerful pull to remove it from the new firm tissue on the surface of the reticulum. In this case also Ohler came to the conclusion that the foreign body was the cause of the paralysis shown some three or four weeks before. The fact that the animal then recovered from the real disease was due to the return of the foreign body into the reticulum. The following grounds are held to justify the supposition:—

- (1) The birth was easy, and no bruising could have occurred.
- (2) The animal stood for two hours after birth was complete.
- (3) The condition of the adhesions is compatible with the idea that these existed at the time of the paralysis.

In a third animal the diagnosis was grounded on symptoms similar to those above described, with the addition, however, of the presence of traces of blood on the fæces. The animal recovered in about ten days, but when it received hay it lay down again, as it had already done two days before, and could not be induced to rise for a further period of thirty-six hours. Under suitable diet, it again entirely recovered. As Ohler had feared the presence of a foreign body, the owner became somewhat anxious, and after an interval of about four months sent the animal to the butcher. The *post-mortem*

examination showed the well-known adhesions, but Ohler was unable to discover the foreign body.

These several cases relate to "paralysis" after calving, but Ohler has seen similar cases before parturition, one of which he describes as follows:—

He was called to a primipara with the history that for some days past she had taken nothing and had ceased to ruminate. On his first examination he found the stomach and bowels not acting, the pulse normal, and the temperature 39.2° C. On pressing over the region of the reticulum the animal groaned and opened its eyes, showing the entire anterior half of the sclerotic.

Two days later the animal lay down, and could not again be induced to rise. Pressure over the reticulum was even more painful than before. On the fourth day after the animal had been down Ohler recommended immediate slaughter. The animal was already thirty-eight weeks pregnant. On opening the carcase he found a sewing needle which had penetrated the diaphragm. The injury caused by the foreign body was quite recent, and no other cause of the continual lying down than this wound caused by a foreign body could be found.

The chief difference between the above described cases of apparent paralysis and those not caused by a foreign body consists in the method of taking nourishment. In all the cases of paralysis Ohler has seen in which there was no suggestion of a foreign body the animals showed unaltered appetite, notwithstanding the difficulty of delivery even to the point of the vagina having been lacerated. In the above-mentioned cases, and especially at their outset, this was never the case. On the contrary, the animals showed symptoms of acute gastric catarrh.

Although foreign bodies may for long remain in the animal's reticulum without producing any bad effect whatever, yet, under the influence of violent exertion, such bodies may suddenly be thrust forward through the diaphragm and even into the pericardium. Similarly, the violent action of the abdominal muscles during delivery, or the forward pressure of the gravid uterus on the organs of digestion during pregnancy, may force foreign bodies through the reticulum, with the results above noted.—(*Wochenschrift für Thierheil*, Nos. 18 and 19, 1902.)

SUPPURATIVE MENINGITIS AS A SEQUEL TO STRANGLES.

Two examples of this rather rare sequel to strangles are recorded by Humann. After an attack of strangles, two seven months old foals showed symptoms of a peculiar disease, from which they died. After extensive abscess formation in the submaxillary lymphatic glands the animals appeared to be recovering. Suddenly, although sensation seemed perfect, symptoms of tetanus set in. The legs were stretched out stiffly, the neck and head were held extended, the ears were immobile. There was, however, no trismus of the muscles of mastication, and oats and hay were eaten with moderate appetite. On attempting to move the animals backwards, however, they fell and seemed unable to rise again. Soon after the appearance of these symptoms a hot painful swelling appeared above the poll, and extended down about one-third of the neck. The head was now held quite stiffly extended, and the animals could only eat when food was introduced into the mouth. The diagnosis of metastasis and metastatic inflammation of the medulla oblongata was formed, and the prognosis was naturally very unfavourable. In a few days the swellings showed fluctuation, and on incision discharged a thin, fluid, ill-smelling, purulent material streaked with blood. On the seventh day after the appearance of these symptoms the animals died.

On *post-mortem* examination a well-marked suppurative inflammation of the medulla oblongata and its membranes was discovered. The remarkable point was that this fatal sequel should affect two foals of the same age at almost the same time and with similar symptoms.—(*Ibid.*, No. 35, 1902.)

THE ORGANISM OF PLEURO-PNEUMONIA.

TARTAKOWSKY and Dschunkowsky repeated Nocard and Roux's experiments on the etiology of pleuro-pneumonia, in connection with an outbreak of the disease which occurred in St. Petersburg during 1900. They also found that the microbes were scarcely visible with a magnification of 1000 diameters. The largest visible bodies, representing masses of microbes, were hardly half a micron in size. They stained best with gentian or methyl violet and with hot carbol-fuchsin solution, but only a part of the micro-organisms were ever stained. The cultures, which are enclosed in collodion envelopes placed in the peritoneal cavity of rabbits, require two to three weeks for growth. In pure cultures the microbe may be propagated for six generations. An injection of the fifth generation into a calf showed its virulence to be slightly lessened. Five camels which were inoculated with attenuated cultures and a calf treated in the same way became immune. The authors declare that the microbe is sufficiently small to pass through the Berkefeldt filter.—(*Ibid.*, No. 37, 1902.)

HEREDITY OF THE ARTHRITIC DIATHESIS: ITS TRANSMISSION TO THREE FOALS BY THE DAM.

By M. DARMAGNAC.

THE mare Orangine, of pure Arab blood, born in 1892 at the Tiaret stud, was subject to colic, and was treated several times for attacks of skin disease. She had three foals, which Darmagnac kept under observation, and of which we give a brief pathological history.

I. *Abandon*, pure Arab, suffered during the entire summer of 1899 from squamous eczema. In 1900 the skin disease reappeared with the onset of warm weather. Without apparent cause cracks occurred around the pasterns, and resisted local treatment. In October the animal showed violent colic of a nervous character, which, however, readily yielded to anodyne treatment (morphine and chloral). In 1901 eczema again appeared at the commencement of summer; cracks formed in the skin; in spite of very careful hygienic treatment the animal had frequent attacks of colic, but these always yielded to anodyne treatment and only to such treatment. Rheumatic lameness set in during training, which had to be discontinued. This animal passed beyond observation after this date.

II. *Badin*, an Arab barb, born in 1899, in 1900 showed patches of eczema, from which the hair was shed. In 1901-1902, during the course of the summer, this animal suffered from generalised eczema and from deep and very obstinate cracks in the skin. It several times suffered from painful colic, which, however, yielded to anodyne treatment, though unaffected by other medication. In April 1902 it was treated for disturbance of circulation, characterised by doubling of the second heart sound and by swelling about the limbs.

III. *Castor*, an Arab barb, born in 1900, in 1901 showed signs of herpes, cracks in the skin, and colic of the same type as that noted in its brothers. In 1902 it showed eczema and cracks in the skin.

The hereditary character of the above conditions in the three animals appears undeniable. The dam seems to have transmitted to her offspring that pathological condition which has been described under the name of the arthritic diathesis, and which is characterised by the complexity of its symptoms. The relations between arthritis, cutaneous eczema, rheumatism in all its forms (affecting both the general muscular system and the muscle of the heart), and enteralgia are beyond dispute. The occurrence of arthritis and its hereditary transmission having been carefully studied in human medicine, in which observation is easier, it appears interesting to cite this observation, which well shows the varying character of the affections arising from the arthritic diathesis in the horse. The three foals showed all the following forms:—

Abandon: Herpes (eczema with cracks in the skin); enteralgia; muscular rheumatism.

Badin: Herpes; enteralgia; cardiac rheumatism.

Castor: Herpes and enteralgia.—(*Revue générale de Médecine Vétérinaire*, 1st February 1903, p. 138.)

THROMBOSIS OF THE ANTERIOR VENA CAVA IN A HORSE.

By Professor ALBRECHT.

On the 5th of June the writer was called in consultation to a carriage horse belonging to Prince Hohenzollern.

History.—The horse (a Mecklenburger), six years old, had shown no signs of disease during the two years he had been in his present owner's possession. The first symptoms were noticed on the 18th of April, and developed as follows: A slight painless swelling at first appeared on the right knee (a few days before a similar swelling had occurred on the left knee, but had again disappeared). The swelling extended upwards towards the right elbow; it remained painless and showed the appearance of oedema. The left knee was swollen. The fore-arm, arm, and left shoulder exhibited oedematous swelling, which extended over the chest region as far as the right scapulo-humeral joint. The swelling increased, extending in both limbs from the foot to the elbow, chest, and sub-sternal region. Finally the two jugular furrows were invaded.

On the 15th May the swelling slightly diminished, but movement was still difficult. The general state of health remained excellent. There was no fever. Nothing could be detected on auscultation or percussion except an increase in the zone of cardiac dulness. No venous pulse, urine normal. Venous stasis appeared in the glosso-facial vessels. The horse became dull and did not lie down. On the 3rd June the swellings were larger than ever, and extended under the abdomen. Appetite failed. On the 5th of June the consultation occurred.

State on Examination.—The animal was in fair condition and the coat brilliant. The swellings were as above described; they were so extensive about the region of the neck that the jugular veins could no longer be raised by compression. The head itself was not swollen, but the veins appeared prominent. The conjunctiva and pituitary mucous membranes were red; the pupil slightly dilated; temperature 38.4° C. The extremities were moderately warm, as were the oedematous patches. Pulse 42, slightly intermittent. The zone of cardiac dulness extended about an inch and a half backwards. The heart sounds were normal. Nothing could be detected on percussion and auscultation of the lungs. Rectal examination revealed nothing. The

digestive apparatus seemed normal. The urine was high coloured, turbid, holding crystals of carbonate of lime in suspension, mucilaginous, and of a strongly alkaline reaction. It deposited little sediment, and contained neither albumen nor colouring substances of a biliary or sanguineous nature. Blood drawn from the jugular appeared normal on microscopic examination. Movement caused the horse extreme pain.

These appearances, and particularly the swelling of the jugular veins and those of the head, suggested some diseased condition of the anterior vena cava, interfering with the return of venous blood. It seemed possible to go even further, and to localise the lesion behind the opening of the axillary veins into the vena cava.

There was nothing indicating disease of the heart (the hypertrophy which existed was not functional). There was no tricuspid insufficiency, no atony of the myocardium, and the pulse was strong. In addition, the normal condition of the heart and of its functions was confirmed by microscopic examination of the blood, which contradicted any idea of hydræmia, leukæmia, or pernicious anæmia. Similarly, microscopic examination and analysis of the urine disposed of the question of disease of the kidneys as a primary cause of the œdema.

The stasis in the circulation being recognised, and the position of the lesion in the vein fixed, it was only possible to regard the case as one of thrombosis of the vessel. As to its nature, the absolutely normal function of the lung disposed of the idea of pathological changes in the bronchial glands, or of an extensive new growth in the mediastinum exerting pressure on the vena cava. Such a tumour developed in the neighbourhood of the vein could not have failed to exercise equal pressure on the trachea, lung, and vagus nerve, and have provoked other symptoms.

The animal, being regarded as beyond hope, was kept under observation for five days longer, during which time its condition became aggravated, and it was then slaughtered.

Autopsy.—Three pints of liquid escaped from the mediastinum. The vena cava an inch in front of the opening into the auricle was surrounded by a swelling the size of a man's fist and of fibrous appearance. Its interior was dotted over with little abscesses containing thickened pus. At this point the wall of the vein was greatly thickened. It contained a thrombus about $4\frac{1}{2}$ inches in length, circular in shape, and red in colour, with a central canal three-eighths of an inch in diameter, but with numerous projections into this canal. In consistence it was dense, firm, and slightly elastic. In front of the thrombus the vein was greatly dilated. The auricle showed several projections of the same character as the thrombus. The other three cardiac cavities and the valves were normal. The heart was slightly hypertrophied.

The formation of the thrombus could be explained in two ways: by compression resulting from the tumour, or by the direct action on the walls of the vein of the micro-organisms contained in the swelling. A bacteriological examination was not made. As to the origin of the lesion, Albrecht was inclined to refer it to strangles, although the horse had shown no symptoms of this disease for two years. The nature of the swelling and the changes in the venous walls, however, were quite in keeping with the view that the disease was of old standing.

The absence of venous pulse during the disease is easily explained by the fact that the tricuspid valves were healthy. The prolonged stasis of venous blood strongly charged with carbonic acid had no more serious effects on the patient than the increase in blood pressure in the aorta. The fact that the nervous centres continued to act normally is explained by their having gradually become accustomed to the new state of things. Thanks to this, the venous stasis produced neither excitation of the respiratory centres nor

acceleration of the breathing movements. The curious fact that the pulse was not increased in frequency in spite of the obstacle to the flow of blood is explained by the regulation of pressure due to the more extensive flow towards the posterior aorta. It is more difficult to explain the intermittency of the pulse, which appears to have been due to nervous atony. There is nothing surprising in the fact that no disturbance occurred in the circulation of the posterior vena cava, pressure in which was not in the least increased. On the contrary, the fact that the anterior vena cava was bringing less blood to the auricle resulted in an increase in circulation in the posterior vena cava. There ought to have been an increase of pressure in the thoracic canal, but not sufficient to produce disturbance. The horse did not show any enlargement of the hind limbs.—(*Zeits. f. Thiermed.*, December 1902.)

THE TREATMENT OF FOOT-AND-MOUTH DISEASE BY A PROTECTIVE SERUM.

By E. NOCARD.

Two years ago M. Jean Dupuy, then Minister of Agriculture, instructed Dr Roux and Professor Nocard to undertake a scientific study of the difficult problem of preventing foot-and-mouth disease. In this laborious investigation the two gentlemen named were assisted by two of their most brilliant pupils, MM. Valée and Carré. For two years they have pursued the subject, but unfortunately have made only modified progress.

The microbe of foot-and-mouth disease has up to the present evaded all attempts at isolation; although it is so abundant in the discharges that the serum of affected animals diluted with 10,000 parts of sterilised water is still capable of conveying the disease, these investigators have neither been able to see it nor to cultivate it outside the living body. That they have failed to identify it is not surprising, for it is so small that it passes through the pores of the densest porcelain filters. It may therefore possibly be beyond the limits of vision. This, however, would not be of great importance if it could be cultivated outside the body and caused to multiply in artificial cultures. Unfortunately, all attempts at culture have remained unfruitful, and this check is the more to be regretted, because when one is master of the specific agent, the microbe of a contagious disease, one has a much better chance of becoming master of the disease itself, of succeeding in attenuating the virulence of the microbe, and thus of transforming it into a vaccine. Even although one fails to produce a vaccine (which is the ideal of prophylaxis), the possibility of obtaining large quantities of virulent cultures makes it easy to prepare a really efficacious serum, either of a protective or of a curative nature. It would be superfluous to recall the remarkable results obtained by serum treatment in diphtheria, bubonic plague, cattle plague, swine erysipelas, and tetanus.

The investigators, therefore, have not attained the principal object of their researches—the discovery or culture of the microbe, which alone would render easy the preparation of a vaccine, or of an efficient serum in quantities sufficient for all requirements. Possibly they will never succeed. One of the first bacteriologists in Germany, Professor Löffler, has for the past seven years been seeking the same object, and up to the present has not found a solution. The efforts of the French investigators, however, have not been completely sterile. They, like Löffler and many others, found that the serum of animals which had recovered from a severe attack of foot-and-mouth disease possessed a certain action on the virus of that disease. Inoculated

in large doses into fresh subjects, it increased to a marked extent their natural resistance against the virus which might afterwards be injected. It diminished the gravity of the disease so produced, and, in cases where the dose injected was sufficient, prevented the disease appearing. But the activity of this serum was so slight that in order to produce good effects it was necessary to inject from 500 to 1000 cubic centimetres.

Like Löffler, they found that, when the immunity of animals which had recovered from a severe attack of the disease was increased by several times injecting large quantities of the foot-and-mouth disease virus, the preventive and curative action of the serum was markedly increased. In this way they finally succeeded in obtaining a serum of such activity that, injected in doses of 20 cubic centimetres into new subjects, it protected them with certainty against the effects of a dose of virus much in excess of that which gave the disease to control animals. These observations repeated many times in the laboratory were entirely confirmed in practice.

Last year, for example, foot-and-mouth disease attacked two animals at the general Agricultural Show on the evening before the close of the show. Fifteen other animals belonging to the same owner, and tended by the same men as those affected, were injected with protective serum. None of these fell ill, though it is probable that several of them already had the germs of disease in their systems at the time of receiving the injection, but the serum prevented the development of these germs. The animals recognised as diseased were in the immediate neighbourhood of others belonging to different owners, the majority of whom consented to submit their animals to the same preventive treatment. Altogether, ninety oxen were injected, and none of these ninety animals developed disease. Since then it has been discovered that amongst the exhibitors ten had foot-and-mouth disease in their establishments some days after the breaking up of the show.

The following may be mentioned: M. F——, on whose farm the disease killed several cows; M. de F——, who lost several animals of value; M. D——; M. G——; M. le Prince de B——; M. M——; M. C——, etc., without counting those of which no reports came to hand. Now, the ninety animals inoculated were distributed in all sections of the show, and were therefore amongst those which afterwards became ill. They were, like these latter, exposed to the chance of contracting the disease, and it seems justifiable to conclude that if they escaped it was in consequence of the serum with which they had been injected. At anyrate, this conclusion cannot be avoided in regard to the animals of MM. M—— and T——, which were looked after by the same men as the two animals discovered to be ill at the show, and the same is equally true of the three cases mentioned below. M. M—— had shown five animals, among them a bull which he thought of selling at Paris. He asked M. Nocard to inject the four others, but not having been able to sell his bull he brought it back along with the injected animals. Hardly had they returned to the country when the bull fell ill, but none of the other four animals contracted the disease.

M. C—— is a milkman carrying on business near Paris. He asked that his cows might be treated, but, after two cows had been injected, yielding to the objections of his neighbours, who predicted all sorts of misfortunes, the least being that the animals would cease to yield milk, he refused to allow the process to be continued, with the result that the ten non-injected cows had foot-and-mouth disease, and that of the whole herd only the two cows which had been treated remained well and continued to give milk. M. C—— was among the first who next year asked that his animals might be treated.

Mr L——, who brought four oxen to Paris, caused them all to be inoculated. Some time after his return foot-and-mouth disease broke out in his

sheds, and all the animals were taken ill except the four which had received the serum.

Finally, we may recount the following case, which occurred during July last. A large breeder, M. C——, had foot-and-mouth disease in certain of his breeding paddocks. Fearing, with reason, that his entire establishment would become infected, he asked that the most valuable of his animals might be treated. M. Carré attended and injected the eighty animals. M. C—— reported the results in the following terms: "I am happy to inform you that up to the present success has been complete. The animals which were treated, although mixed with others which took the disease, have not fallen ill. Foot-and-mouth disease is very bad here. Yesterday and the day before I lost two beasts two-and-a-half years old. Two cows which had been treated but which were in the same lot remained absolutely healthy. The rams which were inoculated have not been affected although all the sheep in the same flock are ill. Of one lot of six bulls, five received a dose of the serum. The sixth, which was left as a control, has alone taken foot-and-mouth disease. My milking cows which I value so highly have resisted. . . . Altogether, of eighty beasts injected none has yet been attacked, although everywhere else on my farm the disease goes on increasing."

Many similar facts might be adduced, but the above will probably suffice to convince our readers of the efficacy and of the innocuous character of the serum. It may be supposed that the difficult question of the prevention of foot-and-mouth disease is therefore solved. Why not apply this treatment, which has given such excellent results, to the whole country? Why not, indeed?

M. Nocard answers the question. No, the problem is not solved. It is in fact very far from being solved, and for the following reason.

The protective serum is undoubtedly very efficacious. Its effect is immediate, but, as in the case of all the serums, the immunity conferred is very temporary, lasting at most a fortnight. After a fortnight, the majority of animals injected may again contract the disease. It will now perhaps be clear why the treatment by serum is not applicable in practice. If one remembers the large numbers of animals which, even in a small outbreak, would have to be injected every fortnight at least with 20 cc. of serum, and will calculate the enormous quantity of serum necessary to satisfy all requirements, there will be no difficulty in understanding.

In the present conditions ruling the production of serum, the matter is clearly impossible. It would certainly become possible although still remaining difficult if we succeeded in obtaining cultures of the microbe of foot-and-mouth disease, but up to the present this is scarcely to be hoped for; and great strides must be made before the process can be extended beyond the smallest and most circumscribed of localities.—(*Revue Vétérinaire*, 1st May 1903, p. 317.)

A NEW METHOD OF REDUCING TORSION OF THE UTERUS.

By E. BACH.

In torsion of the uterus towards the right the upper vaginal folds extend from left to right when passing the hand from behind forwards; the lower from right to left. Provided the conditions are normal, the calf's back will be found in the right flank when the uterus is twisted one quarter, in the middle line of the abdomen when it is twisted one half, and in the cow's left flank when it is twisted to the extent of three-quarters of a rotation. The latter case is very common.

In torsion of the uterus towards the right the cow is cast on her right side,

if possible in the open air and on a declivity, and the head is drawn backwards so that the uterus may be left freer to move on account of the rumen being thrust forward. The cow's head should be held on the ground by a strong man. The feet are not secured but left free, and the casting rope should be loosened. The cow is then turned very slowly on her back by two or three assistants acting under the orders of the veterinary surgeon, who stands near the left flank and manipulates the abdominal region during pauses in the labour pains.

In the case of a three-quarter rotation the foetus can be felt in the left flank or at the linea alba, and during the pauses in the labour pains can quite well be grasped and moved, as the liquor amnii is displaced in the direction of the vertebral column, and the abdominal walls broaden out in the flank region. During the pauses the veterinary surgeon, using both hands, endeavours to thrust the foetus from the left flank towards the middle of the abdomen, and thence as far as possible into the right flank, whilst the two or three assistants, by grasping the feet, slowly turn the cow on to her left side and bring her finally on to her feet. This, however, is not massage of the abdominal walls, but a deliberate attempt to alter the position of the foetus.

As soon as the cow again lies in a normal position or rises to her feet, manipulation is again commenced. On examination one finds the os uteri open or closed, the vaginal folds more or less marked, and the foetus in a normal or abnormal position. Slight variations from normal can be corrected with the hand; greater variations by the ordinary method of turning whilst the foetus is firmly grasped or by extra-abdominal retorsion, that is by manipulation as above described, until, on introducing the hand, the foetus is found to be in a normal position.

The following points must be observed. Firstly, the direction of the vaginal folds must be carefully determined in order to discover the direction of torsion. The animal should not be turned until this has been done. Secondly, the operation should, if possible, be performed in the open air so as to ensure sufficient room, though in case of need it may even be performed in the stall, and in such case it demands somewhat less space than the ordinary method of turning. Thirdly, the cow's head must be kept firmly on the ground by one or two men, otherwise the animal will rise. Fourthly, operation should only be attempted during pauses in the labour pains, when the abdominal walls are relaxed.

The advantages of this method are: (1) It is the best method where the os uteri is closed; instead of having to turn the animal from ten to thirty times, the torsion is often removed on the first or second trial. (2) The membranes are not broken, and the retained liquor amnii greatly facilitates easy delivery. (3) The torsion is rapidly obliterated. The veterinary surgeon fixes the foetus with the hand, and an intelligent assistant may perform the extra-abdominal manipulation.

The writer gives a short résumé of fifteen cases treated as above between March 1902 and a similar date in 1903.—(*Schweizer. Archiv. für Thierheilk.*, XLV Band, 3 Heft, p. 97.)

THE PATHOLOGICAL SIGNIFICANCE OF ŒSTRUS LARVÆ IN THE HORSE'S STOMACH.

By Professor E. PERRONCITO.

The pathological significance of œstrus larvæ in the horse's stomach has been questioned even in recent times by various authors.

It is, however, indisputable that such parasites, though usually regarded as harmless inhabitants of the bowel, may under special circumstances have an

extremely deleterious action on the health of the host. Thus, recent observations have shown that in men appendicitis may be produced by the presence of oxyurides.

The digestive apparatus forms the habitat of an enormous number of microbes, the life processes of which give rise to the production of various poisonous materials; bacteria produce toxins, but anchylostomata, tæniæ, and other helminths also secrete injurious substances. Provided the mucous membrane remains intact, these poisonous materials either fail to be absorbed or are absorbed in such trifling quantity that the animal harbouring the parasites suffers no injury. Should, however, solutions of continuity or epithelial injuries be produced by boring parasites or by other causes, they may pave the way for infections and intoxications of many varying kinds.

Before more closely approaching the question whether such results are brought about in the horse's stomach by the *œstrus* larvæ there found, it is necessary to study the injuries produced by these parasites, in order to bring them into relation with the symptoms which horses thus infected exhibit, and then to recognise the pathological significance of such injuries in various diseases, the immediate or mediate production of which may be referred to injuries caused by such larvæ. Perroncito has made a large number of observations on sections of stomachs prepared by his assistants, Drs Calamida and Lilli.

The *œstrus* larvæ are found both in the right and left half of the stomach, particularly in the latter, and in greater or less numbers, varying from ten up to several hundreds (according to Daubenton and Vallisnieri from 600 to 700) or even up to 1000 or over (Neuman). Sometimes they form a single colony, but frequently two, three, or more colonies are distributed over the surface of the mucous membrane. The young larvæ adhere to the wall of the stomach, bore through the epithelial covering with their shear-like mandibles, and as they develop become more and more firmly attached to the inner surface of the stomach. Usually they penetrate with the head ring into the submucosa, cause disappearance of the epithelial covering and formation of cavities or alveoli with raised convex borders and granulating bases. Not infrequently they bore their way still further in, so that the wound or alveolus contains the second, third, fourth, fifth, and sometimes even the sixth, rings of the larvæ, the tissues receding in accordance with the increasing development of the larvæ. The diameter of the alveolus or space filled by the head and first larval rings may vary between half a millimetre and seven millimetres. The defects appear as simple punctures with a pointed object, or again as large solutions of continuity of circular or rounded, seldom irregular, form.

The base of the wound is also of varying size. It is situated in the submucosa, in which it gives rise to more or less extensive inflammation. The raised edge of the alveolus is formed of hyperplastic epithelium. It sometimes rises like a cone above the mucous membrane and resembles a miniature crater.

At the points where *œstrus* larvæ are adherent the mucous membrane, and at a later stage the entire wall of the stomach, undergoes more or less marked changes, the structure and development of which must be studied, in order properly to understand the symptoms which the infected animals show.

After the young larvæ have become adherent to the mucous membrane, the penetrating or incised openings gradually increase in accordance with their rate of growth. The action of the shear-like mandibles and sharp hairs on the first rings produces an inflammatory point in the submucosa. At the same time the epithelium surrounding the head of the larva undergoes hyperplasia, and the margins of the wound become raised in a circular form. The inflammatory process in the mucosa and submucosa is indicated by the

multiplication of wandering cells, contained in small quantities of connective tissue, the ground substance. That is to say, connective tissue containing large numbers of young cells is formed, and this granulation tissue continually extends laterally and downwards, depending on the mechanical irritation set up by the shear-like jaws and the rings of sharp hairs. At places the infiltration is confined to the immediate neighbourhood of the lesions, but at others the new connective tissue, containing numerous young round cells, extends as far as the centre of the submucosa, and in others again the cellular infiltration or newly formed connective tissue attains the first layer of the muscular coat. In this way hyperplasia of the connective tissue and thickening of the wall of the stomach are produced. In some preparations symptoms of diffuse inflammation between the smooth muscular fibres of the first or innermost muscular coat could be detected, the cells being dissociated and more or less thrust asunder. By pressure this newly formed connective tissue gradually diminishes the function of the muscular fibres, and, later, leads to atrophy and disappearance of the muscular tissue. In many cases the formation of new connective tissue extended to a point between the inner and outer muscular coats, and in these the muscular coats themselves were completely destroyed. Extending from the ulcerated surface, where the submucosa is in direct contact with the armed head and rings of the larva, throughout the entire wall of the stomach may be found masses of this new connective tissue, containing large numbers of round cells. Migratory cells are also found in large numbers in the subserous and serous coats of the stomach. This tissue, in which round cells predominate, is later replaced by fibrillated or fibrous connective tissue, which is poorer in migratory cells but richer in fixed connective tissue cells, so that firm compact masses of cicatricial tissue are thus produced. The localised infiltration, and disappearance of a great part or of the whole of the muscular tissue as a result of these injuries, render the stomach incapable of performing its functions. In such cases the serosa covering the affected parts becomes the seat of a partial inflammatory process, with formation of thread-like or moss-like deposits, which may even be seen on the outer surface of the organ with the naked eye. In one case a portion of the wall of the stomach appeared converted into a hard plate, as though ossified or calcified. Microscopic preparations from this part clearly showed that an infiltration or deposition of amorphous mineral salts had occurred in the destroyed glandular, submucous, and muscular tissues.

Horses affected with *œstrus larvæ* in the stomach or any other portion of the digestive apparatus are more disposed to contract many infectious diseases, a fact easily explained, inasmuch as injurious substances, and particularly micro-organisms, readily find entrance through the numerous small wounds or open surfaces of the stomach or other portion of the digestive apparatus as long as the parasites are in a developmental stage. Even after they attain maturity, and loosen their hold on the membrane, the danger continues, and in fact does not cease until the wound so caused cicatrises and heals. It therefore happens that many horses affected with *œstrus larvæ* become infected with pneumonia. This disease seems particularly to attack horses with *œstrus equi*, and the various forms of fibrinous and septic pneumonia are particularly fatal when infection has taken place through injuries of the stomach, produced by *œstrus larvæ*. Infection with anthrax may also occur by the same channel.

Dr Holil saw repeated colicky symptoms, a temperature of 40° C., and death from septicæmia in a four-year old filly which had probably been infected through the medium of œsophageal injuries caused by *œstrus larvæ*. Neuman has seen cases of perforation of the stomach, produced by *œstrus equi*. In 1897 Dr Conti noted the death of a foal from rupture of the stomach, on the mucous membrane of which 16 larvæ were found, in spite of the fact that the animal had several times been successfully treated with carbon disulphide.

Others have also seen rupture of the stomach caused by œstrus larvæ. Knowing the injurious action which these larvæ are capable of producing, the serious consequences are not surprising. Perroncito has for a long time past collected records of cases of rupture of the stomach which appeared to him due to œstrus larvæ. Quite recently there was sent to the Turin school a horse which, after a journey of over 50 kilometres, had shown symptoms of colic and had died. On *post-mortem* examination, a rupture 6 inches in length was found in the right half of the stomach, towards the small curvature. On the mucous membrane could still be seen traces, where the larvæ had become adherent, of three very extensive colonies of *gastrophilus equi*. Two of these larvæ were still to be found in the left, and one in the right half of the stomach. In addition, extensive injuries were noted in the epithelium of the mucous membrane of the right half of the stomach.

Considering the above facts, which appear to indicate that in horses suffering from œstrus larvæ more or less severe injuries of the stomach may occur, and greater sensibility to infectious diseases exist, more attention should be paid to those methods of treatment by which the larvæ can be removed, and those lasting changes in the wall of the horse's stomach due to this cause can be prevented.—(*Fortschritte der Veterinär-Hygiene*, April 1903, p. 40.)

FOWL PLAGUE.

By Drs OSTERTAG and WOLFFHÜGEL.

In January 1901 a disease of fowls, previously recognised in upper Italy, was conveyed from there, partly directly, partly through the breaking up in February of the Brunswick Poultry Exhibition, and was spread to different parts of Germany, particularly, however, to Wurtemberg, Hesse, Prussia, and Oldenburg. Ostertag and Wolffhügel studied this disease in fifteen fowls which were sent them, and in eighty-eight which were artificially infected. The conclusions they drew from their investigations were as follows:—

The specific organism, which cannot be detected by modern methods of investigation, is contained in the blood, fæces, and nasal mucus of diseased fowls, and is spread by these vehicles. It is killed by a temperature of 70° C. The disease is indicated by dullness, ruffling of the feathers, a sleepy expression, and symptoms of paralysis, and usually produces death in two to four days. It is distinguished from fowl cholera by the fact that it affects fowls and very rarely other birds. Its course is somewhat less rapid, is not attended by diarrhoea, and produces different *post-mortem* appearances, viz. mucus in the nose and pharynx, opacity of the liver, bleeding in the mucous membrane of the digestive and respiratory passages of the oviduct, in the pericardium, and in the peritoneum. In addition, collections of fluid may be found in the pericardial and peritoneal sacs; œdema occurs under the skin of the neck, head, and breast, and in occasional instances the lungs are inflamed. The two diseases may with certainty be distinguished by the discovery of the bacillus avisepticus in the blood where it occurs in large quantities, or by inoculating pigeons, which are only susceptible to fowl cholera. The authors name the new disease "fowl plague."—(*Berliner Thierärztl. Wochens.* 4th June 1903, p. 365.)

INFECTIOUS PNEUMO-PLEURISY OF CALVES.

By Veterinary Surgeon EVERS.

Since Evers three years ago introduced the treatment of diarrhoea in calves by intravenous injections of collargolum, he has been informed by many owners that, whilst the injections prevented the occurrence of the greatly feared diarrhoea, they were succeeded on the ninth to the twelfth day by

symptoms of inflammation of the lungs, from which the animals died two to fourteen days later. At first he was in doubt as to whether the intravenous injections were the cause of the lung disease, but a large number of *post-mortem* examinations of calves' lungs confirmed him in the belief that the cause was really to be sought in the presence of a specific micro-organism. The first suggestion that the cause was specific arose in connection with an estate near the town of Waren, on which no intravenous treatment had been carried out, but on which nevertheless two young calves, aged respectively nine and fourteen days, developed symptoms of inflammation of the lungs and died. In the year 1901 the disease broke out in an epizootic form in D— amongst the breeding calves, of which thirty-four animals died in six weeks from inflammation of the lungs, twenty-one being between twenty days and four months of age. All the animals attacked died.

The symptoms were as follows. The young animals suddenly became dull, sleepy, and coughed frequently. The respirations were markedly hastened, numbering from forty to seventy per minute. The pulse was tumultuous and as high as 100. Temperature 41.5° to 41.6° C. Although the appetite was diminished, the animals usually took their ordinary quantity of milk, but they drank slowly. As soon as the lung disease was completely developed the patient stood with its fore limbs stretched rather widely apart, the head and neck being advanced and the ears laid back. The animal looked dull, breathed with difficulty, and respiration was evidently painful. The nostrils were dilated. The bowels at first acted normally, but afterwards to a less extent. A few hours before death the anus was relaxed, and offensive fluid excreta were involuntarily discharged.

When the calves were from eight to fourteen days old at the time of attack death usually occurred within two to four days, but when of greater age the duration of the disease was from fourteen to twenty-one days. Complete recovery was never seen. In two cases apparent recovery occurred after an attack lasting fourteen days, the subjects being from thirteen to fourteen weeks old. The *post-mortem* examination of these two calves, which were killed at six months old as unfit for breeding purposes, revealed the presence of encapsuled caseous centres as large as a goose's egg in the lungs, and firm adhesions between the opposing layers of pleura.

The *post-mortem* appearance varied according to whether the disease had followed an acute or chronic course. If for any reason one had not seen the lungs, one might have imagined death to have resulted from swine plague.

In animals in which the course of disease had been acute, that is, animals between eight and fourteen days old, the lungs were almost exclusively the seat of change. The apices of one or both lungs were solidified, of a dark red colour, showed numerous pin-point bleedings, and were of firm consistence. Occasionally the apices of the lungs were healthy, and the middle and posterior sections presented a firm, solid, dark-red or brownish-red appearance. The bronchi were filled with froth. From the bronchi of the diseased sections little yellow plugs could be squeezed out. The pleural sacs contained a large quantity of a clear fluid. The bronchial lymph glands appeared sodden and swollen, and in many cases exhibited brownish-red patches on section. In animals in which the disease had taken a chronic course, that is, in animals of six to fourteen weeks old the appearances on section were much more marked. In these lungs the entire gamut of change could be noted, from the commencement of the partially purulent-caseous pneumonia up to almost total necrosis of the lung, with firm union between the parietal and visceral layers of pleura. It was difficult to find even small portions of lung relatively sound. The bronchial and mediastinal lymph glands were often as large as a hen's egg and caseous.

As above stated, *post-mortem* examination reveals the appearances of swine plague in all its stages. And, in fact, the cause of this infectious pneumonia in

calves, which occurs so commonly in Mecklenburg, appears identical with that of swine plague, and it is not improbable that the disease is commonest in places where chronic swine plague is most rife.

Evers considers that the identity of this disease of calves with swine plague is established by the facts given below. In 1901 the greater number of the calves in a certain district died of the above pneumonia, and on *post-mortem* examination Evers had noted the great resemblance between the pathological appearances and those of swine plague. In February Herr V. asked for advice regarding his calves, which were suffering from the disease. One of the young calves was slaughtered, and another, aged 14 days, died after three days' illness. The *post mortem* examination of these animals confirmed the diagnosis. Of the surviving calves five appeared healthy and two had seemed ill for a period of one day. The treatment adopted was founded on the idea that if the cause of the disease was identical with that of swine plague, then swine plague protective serum ought to confer immunity, and might in the early stages have a curative effect. The calves were therefore treated with serum of this character termed Septizidin and prepared in the laboratories of a company at Landsberg. Improvement was so marked that next day the two diseased calves were much brighter, and in a week appeared absolutely cured. The five immunised calves did not take the disease. In 1901 Evers had no further opportunity of making inoculations.

In January 1902 the disease appeared in S. district. Of twenty-eight calves eleven became ill one after the other. Eight died or were killed. On the 16th January the healthy calves each received 10 cc., the diseased 20 to 30 cc., of septizidin. The healthy animals have since shown no sign of disease. Of the three diseased animals, one, which showed no signs of recovering, was slaughtered after some weeks; the two others recovered.

In December 1902 a calf, forming one of a lot of fifty, died of pneumonia. One animal was slaughtered and four became ill. Inoculation with protective serum was performed on the 23rd December, with the result that all save one of the calves treated remained healthy. This one calf had been ill when inoculated, and as it did not rapidly improve it was slaughtered. The left lung contained encapsuled necrotic centres as large as a goose's egg.

Encouraged by these results, Evers in December 1902 sent a fresh diseased lung to the Bacteriological Institute at Landsberg. The result of the examination was as follows: the lung contained bipolar staining bacteria similar to those of swine plague. The virulence of the bacteria discovered was considerable, mice inoculated with 0.1 cc., dying in twenty-four hours; rabbits and guinea-pigs were also killed. The growth on agar and gelatine and in bouillon was similar to that of swine plague and hæmorrhagic septicæmia. In guinea-pigs and rabbits which received intraperitoneal injections there was noted, however, a greater tendency to fibrinous exudate, the liver particularly being coated with a layer of fibrin about 2 mm. in thickness. Cultures obtained from the lung were used as a means of testing acquired immunity. Guinea-pigs which had been protected against swine plague survived the injection of doses of the culture of pneumo-pleurisy of calves several times greater than those which killed unprotected animals; swine plague serum also protected mice against simultaneous inoculation with cultures of the organism. Evers considers this proves the identity of the organism of pneumo-pleurisy of calves with that of swine plague.

Since December 1902 Evers has had excellent results from inoculating calves on the day of birth with 10 cc. septizidin to which a quantity of culture of the pneumo-pleurisy organism has been added. Should the animals become ill, they receive an injection of 20 to 30 ccm. of septizidin. The curative affect clearly cannot be so effectual as protection, inasmuch as the serum cannot restore the injured lung to its normal state.—(*Berliner Thierärztl. Wochensc.*, 23rd April 1903, p. 277.)

VICTORIA VETERINARY BENEVOLENT FUND.

THE fifth Annual General Meeting of the Subscribers to this Fund was held at 10 Red Lion Square, London, on the 3rd June last, when the Council's report for the year was submitted. The Council regard the progress of the Fund as satisfactory, and they feel that there is much cause for congratulation, inasmuch as, although relief to a considerable extent has been afforded during the past year, stock of the nominal value of £500 has been added to the reserve fund. This good work has been for the most part achieved by the offer of a member to subscribe £100 if £500 could be obtained by way of donations from members of the profession. By strenuous efforts this large sum has been collected, and the £100 duly received, thus enabling the Council to add materially to the value of their investments.

The Council regret, however, that owing to the extensive demands made upon the resources of the Society, they have in several cases been reluctantly compelled to refuse aid. They observe that one can hardly realise the extreme cases of poverty which unfortunately exist in the profession, often through no fault of those who apply; shattered health, occasioned either by accident or disease, for the most part conduces to this condition, but every case that is brought to the notice of the Council is fully investigated, and is a matter of individual consideration. For the most part widows of deceased veterinary surgeons are in receipt of relief; in two cases, however, aid is being given to members who are helpless by reason of long continued disease and suffering.

The Council earnestly appeal to subscribers to remit their subscriptions; unfortunately many have not yet done so, with the result that money which might otherwise be devoted to the relief of distress is spent in the endeavour to procure funds. It is felt, however, that there is much cause for general satisfaction, that the fund, having regard to its short existence, has obtained a sure footing, and that with continued efforts on the part of the general members of the profession it will be possible to afford substantial relief in every genuine case of privation. It is in a measure to be regretted that this result has been obtained by the help of comparatively few members only, clearly showing that if the majority of the members would interest themselves to a greater extent than at present, a really great and philanthropic work might be accomplished. It is those who come into direct contact with the many painful and really distressing cases who feel the urgent necessity of an appeal to every member of the profession to help, by every means at his disposal, a society, which has for its sole object the amelioration of the lot of those whose distress is only the more keenly felt by reason of their former social position. A sum of £119 has been distributed by way of relief during the past year, and with real regret the Council have found themselves unable to exceed this limit.

During the past year the sum of £439, 15s. 6d. has been received, made up as follows:—Subscriptions, £69, 4s. 0d.; Life Members, £10, 10s. 0d.; Donations, £323, 3s. 6d.; Dividends on Investments, £36, 18s. 0d.

At the close of the financial year, viz., 31st March 1903, £1225 of Consols and £500 of Norwich Corporation stock were held by the fund. It is hoped that by further efforts this reserve may be considerably augmented during the coming year.

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**A NEW TRYPANOSOMA, AND THE DISEASE
CAUSED BY IT.**

By Dr A. THEILER, Bacteriologist to the Transvaal Government,
Pretoria.

DURING the course of the immunisation of cattle against rinderpest for the purpose of the production of preventive serum, a disease was sometimes met with whose cause seemed to be traceable to the previous inoculation of virulent blood.

The virulent blood infections were made in some instances for the purpose of producing rinderpest and thus maintaining the strain of the virus on the experimental station. In other instances they were part of the simultaneous preventive treatment known as the Turner-Kolle method, used principally for the hyper-immunisation of reputedly immune cattle. The repeated recurrence of the disease led to its investigation, in the course of which it was found that the causal factor was the presence of a trypanosoma.

This disease must have been known already, and my observations lead me to believe that it is common in the Transvaal, perhaps all over South Africa.

I am under the impression that the cattle disease described by Hutcheon, Chief Government Veterinary Surgeon of the Cape of Good Hope, as "Jaundice or Biliary Fever," in his annual report for the year 1897, is identical with that which is the subject of this report. According to him, the disease was first described by Veterinary Surgeon Spreull, who met with it in the Barkly East district, and found it to be of a very fatal character. He noticed it in several cases in herds which had been inoculated with defibrinated blood of cattle immune against rinderpest. In one instance the herd had first been inoculated with bile, followed by virulent blood, and then later with defibrinated

blood from immune cattle. It was observed alike in animals which had been sick with rinderpest and in animals which had not been visibly sick. Hutcheon himself relates that he saw an outbreak amongst cattle on Robben Island, which were inoculated with virulent blood and serum. In this particular instance, out of twenty-six animals attacked, fifteen died, and the evidence proved beyond doubt that the virulent blood produced the disease. Some cows which received serum only were unaffected, the disease being confined entirely to the animals inoculated simultaneously with serum and blood. It was also noticed that grazing on Robben Island had nothing to do with the origin of the disease, as some of the young stock which grazed on the pasture and received no artificial food contracted the disease, as did others which were never allowed out and received only bran and oat sheaves, while others which fed on a mixture of lucerne, hay, and bran also became affected.

I am also of opinion that the disease described by Kolle as "Bovine Malaria," and observed by him at the Serum Station, Kimberley, under circumstances similar to those indicated above, is also identical. A certain abnormal appearance in the blood corpuscles in connection with this disease, which I will allude to later, was noticed by Kolle, but, I believe, wrongly interpreted by him. At the time of the general use of defibrinated blood of salted animals (1897-98) it was repeatedly reported that in some instances cattle were dying from an uncommon disease. A proper investigation, however, was never made.

Nomenclature.

This disease was at that time generally called "gall-sickness" (*Gal-ziekte*). Hutcheon says that the farmers generally attributed it to liver disturbance arising from the dry condition of the veldt.

Gall-sickness is a common and very vague South African term. At various seasons in different localities many diseases are termed gall-sickness, and Hutcheon, who is the leading authority on South African cattle diseases, says this term includes almost every derangement of the liver and digestive organs. In his article on "Diseases of the Liver in some Domestic Animals of the Cape Colony" (*Agricultural Journal*, Vol. XV., No. 1, and Vol. XV., No. 4) he places the following diseased conditions of the liver under the popular term gall-sickness: (1). Congestion or hyperæmia of the liver; (2). Hepatitis or inflammation of the liver; (3). Acute yellow atrophy of the liver; (4). Gastro-duodenal catarrh, catarrhal jaundice; (5). Gastro-enteritis, etc.

I am inclined to believe that the disease in question is embraced in the above list, and may be either of the three first mentioned. Hutcheon says that he is inclined to attribute the origin of acute yellow atrophy of the liver to some special cause.

Inasmuch as a disease like redwater, caused by intracorporeal parasites, and accompanied by congestion, jaundice, fatty and albuminoid degeneration of the liver, and even necrosis of the cells of that organ, is often mistaken for a primary liver complaint, it is obvious that a false interpretation of other diseases, caused by hitherto unknown blood parasites which produce a similar pathological effect, may easily occur, and that the lesions observed in a morbid liver are readily taken to be the originating cause. It follows, therefore, that the term "gall-sickness" indicates principally those diseases which are accompanied

by derangements of the liver, the original cause of which, however, has not necessarily to be looked for in that organ.

The Transvaal farmer's knowledge of the disease which he terms "galziek" (gall-sickness) is ancient history. He is familiar with the process of "salting" (as he calls it) or immunising his oxen, by acclimatisation or inoculation; but when talking of immunity he is *au fait* with all the details, so that he certainly cannot have a disease in his mind which might be caused by bad food, dry veldt, or poisonous plants, although he may fail to provide an explanation. On the other hand, South African redwater is easily and often mistaken for gall-sickness, and the two diseases are commonly looked upon as identical. There exists immunity against redwater, and, owing to the popular confusion of the two diseases, the observed immunity may have been mistaken for that against redwater. Moreover, the expression gall-sickness has existed in the Transvaal since 1871, before redwater was known there, and gall-sickness now exists in those parts of South Africa in which redwater is unknown.

I am under the impression that the disease observed by me to be due to the presence of a trypanosoma is a form of gall-sickness, and is probably the disease which is known in the Transvaal by that name.

Geographical Distribution.

If we interpret the term gall-sickness to mean trypanosoma, then, according to the reports that appear from time to time in the different Agricultural Journals and Newspapers, the disease is all over South Africa. Complete acceptance of this view depends, of course, upon the evidence in the possession of the Cape and Natal experts, and whether they are thereby able to place the disease caused by the trypanosoma under the heading which I claim for it. I have met with the trypanosoma in cattle which must, in my opinion, have brought it with them from the Zoutpansberg district (north of the Transvaal), from the Komati Valley (east), from the Standerton district (south), from Klerksdorp (west); also in cattle, coming from the Cape Colony and the Orange River Colony. I have found it in a Madagascar ox which stayed some time in Natal before arriving in the Transvaal. I also found it in a sample of blood taken from a sick ox in the Carolina district in the High Veldt. These observations indicate that the trypanosoma is generally distributed, and that climatic and geographical conditions have very little to do with its occurrence.

Gall-sickness is generally reported to be prevalent at the beginning of and during the warm season, and this fact coincides to some extent with my observations in regard to the disease caused by the trypanosoma. As the disease is, however, an inoculable one, and very often transmitted by the process of immunisation against rinderpest, I have observed it all the year round in cattle which had been thus treated.

Cause of the Disease.

As already indicated, the inoculability of the disease from a sick into a healthy susceptible animal led to a search for the causal parasite. This was found in the form of a trypanosoma. Bruce of London, and Laveran of Paris, to whom I sent specimens, declared

it to be different from any hitherto discovered, and gave it the name "*Trypanosoma Theileri*."

This flagellated parasite resembles in general appearance the allied parasites of the tsetse disease in South Africa, the Mal de Caderas of South America, the Surra in India, and la Dourine in Algeria. It may, however, be distinguished at once from any of the above mentioned parasites by its larger size and by its polymorphic character.

The quickest method to find the trypanosoma is to make the examination on the living blood, it being sometimes present only in small quantities. A disturbance of the red corpuscles by being whipped in all directions leads to its discovery. In a preparation of fresh blood the activity of the parasite is so great that its form can seldom be recognised directly. In preparations kept for some hours, even up to two days and longer, the trypanosoma may still be found alive, and, its agility having become less, the form and nature are easily recognised. It moves exactly as the trypanosoma of the tsetse, mostly with the flagellum in front, but frequently also with the opposite end. When the movements become slower a long flagellum can be noticed, and the undulating membrane then becomes quite visible.

In stained preparations the structure of the parasite is shown with much clearness. The usual aniline dyes used for the examination of blood films give for diagnostic purposes a fairly good result, but the structure is best shown by any of the modifications of the Romanowsky method. I have obtained equally good results by Laveran's method, also by staining according to Muller's indication for human malarial parasites. Azur 1 and 2 give good results, when the quantity of any of the azurs is taken in proportion of 1:5 eosin, and after a contact from two to three hours with the colouring mixture. I failed completely to stain the flagellum with Reuter's eosin and methylene-blue, although the centrosoma and nucleus are sometimes very clearly stained.

In such preparations the body of the parasite stains a dark blue; the nucleus, the centrosoma, and the flagellum a deep red to a violet colour.

Two forms of parasites may be observed, the distinguishing feature being the position of the centrosoma. In the ordinary form the centrosoma lies at one end (the posterior) of the body, a considerable distance from the nucleus; in the other rarer form it is quite near the nucleus, and is occasionally seen attached to it, or lying actually on it.

The length of the parasite varies between 20μ up to 70μ , and the breadth from 2 to 6μ . The longest parasites are usually found amongst the ordinary forms, and the broadest under the rare forms.

There may be some doubt whether these forms belong to one and the same species, but experiments lead to the acceptance of the view that they do. I base this view on the observation that the injection of blood which contained the rarer forms of the parasite into susceptible cattle produced the ordinary forms in subsequent course. Further, it can very often be noticed that in one and the same animal both forms are present, and also intermediate forms in which there is a variation of the distance between the nucleus and the centrosoma;

so it may be accepted that the rarer form is a stage of development of the commoner form. I was unable to reproduce the disease with either of the two forms in any animal except cattle. It is, however, certainly a striking peculiarity that the rare form was noticed in one ox exclusively.

Laveran, to whom I sent specimens of the two mentioned types, describes the one (ordinary form) as *Trypanosoma Theileri*, and the rarer one as *Trypanosoma Transvaaliense*. ("Comptes rendus des séances de l'Académie des sciences." Tome cxxxv., p. 717.)

As indicated above, I have observed that the injection of blood which contained the rarer forms, which Laveran described as tryp. *Transvaaliense*, produced in the injected animal the ordinary form; hence the interpretation that the trypanosoma may be polymorphic.

The ordinary form is the typical trypanosoma, and it may be regarded as a larger edition of the trypanosoma *Brucei* in so far as its morphology is concerned. The nucleus is fairly large, usually spherical, oval, or dumb-bell-shaped, and occasionally spindle-shaped. The centrosoma is very distinct, and from it, but separated by a clear zone, starts the flagellum, which lines the undulating membrane and goes over in a long whip. The protoplasma is finely granular, the granules taking the stain very deeply when the blood has been kept for some hours before the smears are made.

The rarer form is, as already mentioned, broader, the nucleus shows greater dimensions, and is, in the majority of cases, less compact than in the ordinary form. The body in many cases does not exactly show the common trypanosoma shape, so familiar in tsetse-disease, but is usually more contracted, and shows abnormal shapes—round, oval, lacerated, etc. The centrosoma is very clear, and takes the stain easier than any other part of the parasite. It is usually round, but frequently elongated or rod-shaped.

Mode of Reproduction.

The ordinary forms divide as Laveran and Mesnil have described for trypanosoma *Brucei*, namely longitudinally. The centrosoma divides first, followed by the splitting of the flagellum. I have, however, frequently met with forms in which two nuclei were present and only one centrosoma and one flagellum.

In the rarer forms the character of the longitudinal division is not so easily demonstrated. The protoplasmic body appears to have a spherical or oval shape, in which posterior and anterior ends are hardly discernible. Then, in some forms two centrosomes are recognised, attached to one nucleus, and in a further stage the nucleus may be found enlarged and drawn out. Then, again, a stage is met with wherein one body, two nuclei, and two centrosomes are seen, separated from each other, and lying in a protoplasmic mass. In a still further stage the protoplasma begins to separate. It is impossible to discern whether such forms represent a longitudinal or a transversal division. The flagellum follows the centrosoma in its division. As already mentioned, I have seen the centrosoma at different distances from the nucleus; the furthest in the ordinary form, the nearest in the rarer form. Then, again, it is not uncommonly found sitting directly on the nucleus. This phenomenon leads me to think that the centrosoma wanders from the far end to the nucleus when the parasite

prepares itself for multiplication. This would explain the polymorphic character of the trypanosoma.

Abnormal forms are commonly met with which must be explained either as degenerated or mutilated parasites, seeming to be of very fragile structure.

Involution Forms.

The trypanosoma seems to be easily killed. Involution forms are met with in nearly every preparation. It is the protoplasm which first loses its power to retain the stain, then its contours become indefinable, and the parasite, as such, is sometimes only recognisable by the deeply stained centrosoma, the flagellum, and the nucleus. The latter gradually flattens out, takes the stain but faintly, and finally can no more be recognised. Centrosoma and flagellum seem to offer the most prolonged resistance, and are frequently met in a free state between the red corpuscles.

Agglutination.

In preparations freshly examined, and also in those stained, the phenomenon of agglutination, as described by Laveran and Mesnil in



Trypanosomata showing agglutination.

their paper on "Tryp. Lewisii and Tryp. Brucei," is but seldom observed. Occasionally two trypanosomata with their posterior ends adhering together are met with. When, however, the defibrinated blood which contains the parasites is left alone for some time (over night) the trypanosomata come to the surface and adhere together in the form of rosettes with the posterior ends hanging together. I also met with this phenomenon in blood after adding serum of a calf which had repeatedly been inoculated with blood containing trypanosomata. The phenomenon, however, only occurred a few hours after the addition of the agglutinating serum.

In one case (Calf 4) where the trypanosomata were in enormous quantities agglutination was observed in the freshly examined blood twenty-four hours after the animal had received a large quantity of immunising serum (200 cc.).

Previous to this injection the same blood was repeatedly examined, and the agglutination was never observed, so that the phenomenon seemed to be due to the injection of the immunising blood.

Vitality of the Trypanosoma.

The parasites live in defibrinated blood in test tubes up to seven days when kept at the temperature of the room or in the ice-box, but not so long when placed in the incubator. It seems that when the blood in the incubator becomes laked the parasites soon die.

Defibrinated blood containing trypanosomata was diluted with horse serum, and with physiological water, and it was then found that parasites lived as long therein as in undiluted blood. Defibrinated blood mixed with peptone bouillon showed the living parasites for a little over forty-eight hours. The addition of ordinary water, glycerinated water, and normal saline solution which contained only .5 per cent. carbolic acid, killed the trypanosomata quickly, and destroyed them so that no trace was left of them.

Exposure to the temperature of 50° C. kills the parasite within twenty-four hours.

The injection of blood which contained living trypanosomata whose motility was reduced so far that the flagellum seemed to be the only living part did not prove to be infectious in the two experiments made.

Associations with other Parasites and Diseases.

The trypanosoma is very frequently seen together with pyroplasma bigeminum of ordinary South African redwater, and also with the bacillary form of the same parasite, which is the cause of the virulent redwater (Rhodesian tick fever).

I have frequently met with cases in cattle suffering from rinderpest, and in one case I found the parasite associated with a spirillum. The rôle of the latter as a cause of disease is not yet ascertained. It is often very difficult to know to which of the parasites the lesions in a dead animal should be attributed.

Pathogenic Effect of the Trypanosoma on the Blood.

The different results of injection with trypanosoma among cattle will be demonstrated hereafter, and the change effected in the red corpuscles by its influence will also be described. In the first instance anæmia is produced, which may be slight, acute, or very grave, the number of red corpuscles becoming reduced to a very small quantity per cmm. In a light infection the numbers of white corpuscles are scarcely increased. Where, however, the trypanosomata are frequent, the white corpuscles seem to increase in direct proportion. The eosinophile cells are usually observed to increase in number, and are sometimes very numerous.

Inoculations.

Injections of defibrinated blood containing the trypanosoma were made into three horses, nine dogs, six sheep, two goats, twenty rabbits, fifteen guinea-pigs, five rats, and one mouse. The material for the inoculation was taken exclusively from cattle which were spontaneously infected. None of these inoculations produced the parasites in the infected animals, and none died from the effect of a trypanosoma infection. In some of the sheep a febrile reaction took place after the injection, the cause of which could not be determined. Trypanosomata were never seen in their blood. Knowing by experience in connection

with experiments with trypanosoma Brucei that sheep die with nagana without ever showing a single parasite in the blood, the inoculation of large quantities of blood of these sheep (150 cc.) was made into two calves without producing the parasite.

The conclusion may therefore be drawn that the trypanosoma only infects cattle.

Manner in which the Disease is Spread.

In reference to the analogy of the nagana disease, which is carried by the tsetse fly, it may be assumed that the new trypanosoma infection is also carried by winged insects. A search for blood-sucking flies was made, when it was found that a hippobosca is pretty frequently met with in cattle, and by preference often settles in the region between the hind-legs, but may be found running all over the body. Such flies were placed in cages made for the purpose, and put on animals infected with trypanosoma. It was found on dissection of such a fly, which had been feeding in the way indicated, that the blood in the stomach contained living trypanosomata, which, up to one hour after their removal from the infected animal, could be found as agile as in the blood of the animal itself. These examinations were repeated several times, and animals were selected in whose blood the trypanosomata were in varying quantities. It was found then, as might have been anticipated, that the quantity of parasites in the fly's stomach was in direct proportion to the quantity the animal's blood contained.

Being convinced that the hippobosca can contain living trypanosomata for at least one hour (it has not yet been tested how long the trypanosoma keeps its vitality in the fly's stomach), I decided to try the infection of cattle by the medium of the fly's bite. For this purpose some flies were kept over-night in order to make them hungry, and were then placed on the groins of an infected calf. To give the experiment every chance of success, the place where the flies were put to feed was first shaved, as was also the spot on a clean animal where they were placed for infection. Feeding by turns on a sick and on a clean animal was thus repeated several times, in order to secure an infection. Out of four experiments made in this way, two were successful. It must be stated here that the experimental animals were kept together with control animals in a stable, to exclude spontaneous infection, and that none of the control animals showed a spontaneous infection.

The incubation periods coincided typically with the period which is observed after artificial infection with small quantities of virus. The infection was only a slight one, and thus shows also that the presence of trypanosomata in the blood does not necessarily lead to clinical symptoms, and may therefore be easily overlooked in ordinary life. I consider it to be proved that the hippobosca is a medium of the propagation of the disease, but it is quite possible that other blood-sucking flies, such as tabanidæ, may be carriers as well. Tabanidæ are, however, not so numerous, and I have not had an opportunity to experiment with them.

I sent two specimens of the hippobosca with which I made the successful experiments to Dr Laveran, who had them identified by Dr Speiser of Bischofsburg. One of them was declared to be

hippobosca rufipes (v. olfers), and described as a common parasite in South Africa; the other one as *hippobosca maculata* (Leach), a common fly of India, which Speiser thinks has only lately been introduced to South Africa, probably by military horses from India. ("Comptes rendus hebdomadaires des séances de la Société de Biologie," Tome lv., No. 7, 1903.)

Description of the Disease.

The presence of trypanosomata in the blood of an animal may give rise to a disease which reveals itself by symptoms varying in severity in different animals. I therefore make a distinction between an acute and a subacute disease. At this juncture it is perhaps advisable to describe several typical cases.

I. Acute disease, with pathological change of the red corpuscles.

Ox 185, about two years old, and weighing 390 lbs., was inoculated on 15th November 1901 with 5 cc. virulent blood from No. 1 S.A.C. and 20 cc. serum No. 1 to test the strength of the serum, the dose being in proportion of 30 cc. to about 600 lbs. live weight. The temperature rose in the evening of 20th November to 105 degrees, and the animal was in fever for four days, after which the temperature subsided to normal. There were absolutely no other symptoms. On the 9th December the temperature began to rise again, diarrhœa followed, and the animal died on the sixteenth day while it was being bled for the purpose of examining the blood, which was found to be watery and contained enormous quantities of nucleated and basophile erythrocytes.

Post-mortem was performed immediately after death. General anæmia and slight jaundice. Lungs collapsed, very pale, but otherwise normal. Pericardium distended with yellow serum. Epicardium œdematous. Heart muscle flabby and petechiæ on apex. Endocardium normal. Liver slightly jaundiced, friable, and bloodless. Bile dark green and thickish. Spleen enlarged, the pulp firm, but of a brownish colour, and the trabeculæ very marked. Kidneys pale, capsule œdematous. Bladder full of yellow urine. Stomach contained softened food. The mucous membrane of the fourth stomach was normal; that of the jejunum and ileum yellow and slightly swollen. The same appearances were present in the colon and cæcum. The serosa of the whole intestinal tract was œdematous, especially along the mesenteries. All the intestinal glands noticed were enlarged. The flesh was slightly brown in colour. Examination of the blood showed poikilocytosis, megaloblasts, and normoblasts; besides, the cells contained basophile granulations. No trypanosomata were found.

Two calves were inoculated with blood from this animal, viz., No. 26 and No. 29, each 20 cc. Calf No. 26 showed no result from this inoculation. Calf No. 29, however, gave a slight reaction. The blood was examined during the reaction, and an increase of eosinophile cells was observed, but after the subsidence of the reaction the presence of the trypanosoma was demonstrated.

Ox D.—An aged animal, weighing 1135 lbs., was inoculated on 8th July 1901 with 1 cc. of rinderpest blood drawn on 1st July at Maseru. Although the injections were repeated, and the aggregate amount on 15th October amounted to 5775 cc., the animal did not contract

rinderpest. It was probably immune, either on account of inoculation, or as the result of a previous attack in 1897-98. During the same period it was bled for serum, the quantity drawn amounting in all to 2000 cc. On 14th December it received an injection of 1500 cc. virulent blood, and on the 16th its temperature began to rise. At first this was thought to be the usual reaction of the injection of virulent blood, but the temperature remained high until 1st January 1902. The animal lost appetite, and during three days did not ruminate. It was usually found lying down, or, when on its feet, with hanging head and drooping ears. It was constipated, passing only a little dry dung. The animal gradually recovered. On 20th January, a few days before the temperature became normal, the blood was examined, and was found to show poikilocytosis, nucleated cells, and basophile granulations. On the 30th the blood was again examined, and the basophile cells were in increased numbers. On the 1st January the red cells with basophile granulations numbered from fifteen to twenty in each field, and the nucleated erythrocytes from six to ten. The blood corpuscles were counted on the 2nd January, giving 2,500,000 per cmm.

3rd January. Basophile granulations still present, erythrocytes 2,900,000 per cmm.

4th January. Nucleated and basophile cells still present, blood count 3,300,000.

5th January. No nucleated erythrocytes; basophile granulations decreased; blood count 3,810,000 per cmm.

6th January. Basophile granulations rare, not more than one in from two to three fields. Blood plates frequent; polynuclear cells more frequent than before; blood count 4,500,000.

7th January. No more granule-bearing cells to be seen; red cells 4,690,000 per cmm.

During the following days the blood became normal, and the red cells numbered 5,150,000 per cmm. The examination was then discontinued.

On the 28th December 1901 the ox was bled, and 40 cc. of the defibrinated blood injected into the jugular of Calf No. 25. A reaction took place after four days, the temperature rising on 6th January to 106° F. The blood was examined during this reaction. Nothing particular could be found, but on the 13th January, or sixteen days after the injection, trypanosomata were seen. In this animal a considerable loss of flesh and slight indisposition was noticed, but the blood showed no pathological changes. The trypanosomata disappeared from the blood on 20th January.

Calf 53.—3rd July 1902. Heifer calf, one year old. This calf, bought for the purpose of producing calf-vaccine and recently arrived from the Orange River Colony, was only a few days in the stable attached to the laboratory when it was noticed to be very ill. Its head was hanging, the ears drooping, the flanks sunken, the gait staggering, and the animal was lying down most of the time. Food was refused and rumination ceased. The temperature in the evening was 104.6° F., but only 99.8° the following morning.

Examination of the blood showed a very pronounced poikilocytosis and pretty frequent erythrocytes with basophile granulations. No trypanosomata could be seen, nor any other blood parasites. The

animal improved within the next few days, took food, and started rumination. Nevertheless it lost condition rapidly, and was accordingly killed on the 16th July 1902.

Two days previously the examination of the blood showed the presence of trypanosomata. The *post-mortem* was made directly after slaughter, the animal having been bled to death.

There was a serous infiltration of the subcutaneous tissue of the breast, abdomen, and shoulder. General anæmia very pronounced (due to bleeding). There was a mass of organising fibrous tissue in the pleura of the right apex (remains of pleuro-pneumonia), and some slight oedema of these organs. The pericardial fluid was increased, the spleen enlarged, and the pulp softened. The liver seemed to be normal in colour, but the bile ducts contained much bile; kidneys very pale in colour and oedematous, and the calyx had a gelatinous appearance. The mesenteric glands were enlarged. The intestinal tract showed no lesions. Microscopical examination of smears from the internal organs proved the absence of trypanosomata, although they were pretty frequent in the circulation. There was no change in the red corpuscles as mentioned on the day of first examination.

The tabulated record (p. 204) showing experiments on seventeen animals is interesting, as the disease was probably due to one and the same cause, and in the course of the disease similar phenomena could be observed in every case. The appearance of trypanosomata was, however, in most of the animals complicated with the appearance of pyroplasma bigeminum, and, as will be shown later, the appearance of basophile cells is, in my opinion, not exclusively pathognomonic for the trypanosoma.

It will, however, be noticed that, while in some instances the pyroplasma was absent, the basophile granulations were nevertheless present, together with the trypanosoma (Cases 2, 13, 14). In other cases pyroplasma alone were present and no basic cells (Cases 15, 16), and in one case trypanosomata and pyroplasmata were present and no basic cells.

The history of the outbreak is as follows: The animals were short-horn heifers, most of them in calf; they were imported from Argentina and were brought directly from the coast to Pretoria, where, shortly after their arrival, they were treated for rinderpest by the simultaneous method on 16th August 1902. The virulent blood was taken from an ox (HD), which blood on microscopical examination proved to be free from any parasite; also the *post-mortem* of the Ox HD showed nothing but typical rinderpest.

All the animals went through a typical reaction of rinderpest, but the febrile reaction did not stop after the typical time, and a second reaction followed shortly afterwards, which led to the examination of the blood. The result is shown in the accompanying table.

The *post-mortems* of the animals which during life had trypanosomata, basic cells, and pyroplasma differed very little.

Case No. 1.—This animal showed pyroplasma only on the first microscopical examination. In the course of the disease it became very emaciated, and five days before death was unable to rise. It was accordingly killed, and the *post-mortem* made directly after death. The flesh had but a slightly jaundiced colour. The lungs showed hypostatic oedema; the endocardium of the left ventricle was thickened

TABULATED RECORD A.—SHORTHORNS 1-16 AND CALF 4.

No.	20/9/02	2/9/02	4/9/02	5/9/02	8/9/02	10/9/02	13/9/02	
1	Trypanosoma and pyrosoma	Tryp. Basic cells. Nucleated cells. Two pyros.	Tryp. Basic cells	Tryp. Basic cells rare	Tryp. Basic cells	Tryp. Basic cells rare	Tryp. No basic cells	Killed 18/9/02
2	Basic cells	—	Tryp. Basic cells	Tryp. Basic cells rare	Tryp. Basic cells	Tryp. Basic cells rare	Basic cells rare	Tryp. No basic cells
3	Tryp. and pyros.	—	Died 3/9/02	—	—	—	—	—
4	Tryp. and pyros.	Tryp. Basic cells	Tryp. Basic cells	Tryp. Basic cells	Tryp. Basic cells	Tryp. Basic cells rare	Tryp. No basic cells	—
5	Pyros. Basic cells rare	Tryp. Basic cells	Tryp. Basic cells. One pyrosoma	Tryp. Basic cells rare	Tryp. Basic cells	Tryp. Basic cells	No basic cells	No basic cells
6	Basic cells	Tryp. Basic cells. Pyrosoma rare	Basic cells. Polychr. nucleated cells	Basic cells. Polychr. nucleated cells	Basic cells	Basic cells	Basic cells rare. Nucleated rare	No basic cells
7	—	One pyrosoma	Basic cells. Nucleated	Tryp. Basic cells. Polychr. Nucleated cells.	Died 6/9/02	—	—	—
8	Pyrosoma	Tryp. Basic cells. Two pyros.	Tryp. Basic cells	Pyrosoma	Tryp. Basic cells	Basic cells rare	Basic cells	Basic cells rare
9	Pyrosoma	Died 29/8/02	—	Tryp. Basic cells rare	—	—	—	—
10	Basic cells	Tryp. Basic cells. One pyrosoma	Tryp. Basic cells	Tryp. Basic cells rare	Trypanosoma	Tryp. Basic cells rare	One Tryp. No basic cells. One pyrosoma	—
11	—	Trypanosoma	—	—	Basic cells	Basic cells frequent	Basic cells	Died 23/9/02
12	Pyros. Basic cells	Died 29/8/02	—	—	—	—	—	—
13	—	Trypanosoma	—	Basic cells rare. Polychr. nucleated	—	—	—	—
14	Tryp. Basic cells	Tryp. Basic cells. Nucleated cells	Tryp. Basic cells. Polychr. nucleated cells	Tryp. Basic cells numerous	Basic cells	Basic cells	Basic cells rare	—
15	Pyrosoma	Died 27/8/02	—	—	—	—	—	—
16	Pyrosoma	Died 28/8/02	—	—	—	—	—	—
Calf 4	—	Tryp. very numerous. Pyrosoma	Tryp. Basic cells. Nucleated polychr. Pyrosoma	Tryp. Basic cells. Pyrosoma	Tryp. Basic cells. Pyrosoma	Died 9/8/02	—	—

and white (chronic lesion). The liver was normal. The spleen slightly congested. The kidneys slightly yellow in colour. The urine normal, and the intestinal tract did not show any pathological changes.

Case No. 3.—Died on the 3rd September 1902. *Post-mortem* five hours after death. Rigor mortis present. A general icterus was slightly pronounced. There were broncho-pneumonic lesions in both lungs. The liver was jaundiced, and the gall-bladder contained greenish bile. The spleen was normal. The kidneys of a somewhat brownish appearance, and the urine clear. There was a general catarrhal condition of the mucous membrane of the intestinal tract.

Case No. 7.—Trypanosomata and pyrosomata were present in the blood. Died on the 6th September 1902.

Post-mortem shortly after death. Very pronounced icterus was present; the fat especially was quite yellow. There was a broncho-pneumonia in the right lung, with collateral œdema and also emphysema. The colour of the surface of the epicardium was brick-red from confluent hæmorrhages. The liver was enlarged and yellow; the bile thickish and of a gelatinous nature. The kidneys pale. The urine brown, and when boiled formed a deposit. The intestinal tract was normal. Round pyrosomata could be found in smear preparations from the liver, spleen, heart, and kidneys.

Cases No. 9, 12, 15, 16 were typical cases of redwater.

Case No. 11.—*Post-mortem* seventeen hours after death. There was jaundice; enlarged spleen; yellow, soft, and friable liver. The other organs were normal.

Relation of the Presence of Trypanosoma to the Occurrence of Basophile Granulations.

In the first mentioned cases (188 Ox D) no trypanosomata were observed in the blood when examined by the microscope; their presence, however, was proved by injection of certain quantities of the same blood into susceptible animals, whereupon the trypanosoma appeared in the blood of the injected animals. Most of the cases in the Argentine cattle were complicated with redwater parasites (mostly in a slight degree only), and the occurrence of basic cells might just as well have been due to the presence of pyrosoma alone, as will be proved later. Only the case of Calf 53 proved the occurrence of both trypanosomata and basophile cells to which no objection could be raised. It will therefore be necessary to prove by experiment that the trypanosoma alone is capable of producing the mentioned pathological change of the red corpuscles.

Experiments.—Calf 49 was injected with 50 cc. blood of Calf 47, taken in the reaction, during which trypanosomata were present. Basophile granulations and endoglobular parasites were absent. The basic cells appeared six days after injection, and the trypanosoma six days later.

Calf 32, which to previous injections of trypanosoma blood proved immune, received 1000 cc. of blood of Calf 49 (for hyper-immunising purposes). Trypanosomata appeared on the fifth day, basic cells on the sixth day, but disappeared again a few days later.

Calf 54 was also injected simultaneously with 50 cc. blood of Calf 47, which blood only contained trypanosomata. Trypanosomata made

their appearance seven days after injection, and were present during thirteen days. Five days later the basophile granula in the red corpuscles appeared.

Ox 61 was inoculated with 50 cc. blood of Argentine shorthorn heifer No. 2, in whose blood, as will be noticed in Table A, no pyrosomata were found. Five days after injection parasites made their appearance, and seven days later the basophile granulations could be noticed, and they were still present some time after the disappearance of the trypanosoma.

Calf 9. This calf had already been injected with blood containing basic cells but no visible parasites, and had had a reaction as the result of such injection, with the occurrence of parasites. It was again inoculated with 100 cc. of blood of a calf which contained only trypanosomata. This injection was followed by a reaction, during which nothing particular could be seen in the blood; and after the termination of the fever basophile cells appeared. The number of red corpuscles amounted that day to 2,420,000 per cmm. The animal was ill, refused food, and stopped rumination, but made a good recovery. No trypanosomata were found during or after the reaction.

From the foregoing notes it may be deduced that the trypanosomata are responsible for the production of the basophile granulations. It will be noticed from the few cases in which the red corpuscles were counted that the anæmia was very pronounced in those cases in which the basophile granulations appeared.

Basophile Granulations not Typical for the Trypanosoma Infection.

Although at one time it appeared to me that the formation of basophile granulations in the red corpuscles was typical of trypanosoma infection, further evidence led to the opinion that the infection with the pyroplasma exclusively may also have the same result.

Ox 45 (Cape ox), nine years old, was immunised against rinderpest. Ten days after the simultaneous injection of serum and virulent blood red urine was observed, and the microscopical examination proved the presence of typical pyrosomata (big oval forms). Four days later basophile granulations were already present. The animal made a good recovery, and repeated examinations of blood proved the absence of trypanosomata.

Ox 347, Cape animal, two years old. The same history as before. Red urine was noticed eleven days after the injection of serum and virus, and five days later basophile cells, nucleated red corpuscles, etc.

Ox 336, Cape animal, four years old. Red urine was noticed twelve days after injection, and the oval pyrosomata were present for some time. Seven days later basophile granulations were noticed. No trypanosomata were found. Four fresh animals were injected with blood from this ox. This produced redwater in one ox, but trypanosomata could never be found.

Ox 261, Cape animal. Simultaneously treated for rinderpest 2nd October 1902. On the twelfth day red urine appeared, and the pyrosoma bigeminum was seen in the blood. The ox died four days later. *Post-mortem* was made immediately after death. There was a very pronounced icterus, the blood had a brownish hue, and left but a faint stain. The lungs were emphysematous, the epicardium brick-red,

the endocardium yellowish. The liver was enormously enlarged and icteric; the bile brown, with a lot of sediment; the spleen very large and softened. The kidneys had a brownish colour, and the urine was red. The lymphatic glands were slightly enlarged, the stomach and intestines in a catarrhal condition. The blood contained pyrosomata, numerous granulations, and nucleated cells. This animal was bled on the day when red urine was noticed, and pyrosomata were seen microscopically. A certain quantity of this blood was injected into three oxen (already immune for redwater) with negative results.

From the above notes it would seem to be proved that infection with pyrosoma bigeminum can produce basophile granulations. So the conclusion may be drawn that the formation of these granulations is a sequela of the acute anæmia which is caused either by the presence of pyrosoma or trypanosoma. Basophile cells are therefore an index of infection with either of the two parasites. But, as the parasite itself is not always present, the exact diagnosis may be impossible.

The Basophile Granulations.

The acute disease caused by the presence of trypanosoma simulates anæmia of an acute type. There is destruction of the red corpuscles, poikilocytosis, microcytes and macrocytes, normoblasts and megaloblasts, and basophile erythrocytes.

In veterinary pathology all these different conditions have been described, except the presence of basophile granulations in the red corpuscles. These granulations are easily seen in smear preparations stained with methylene-blue (Kuhne's blue), when the red blood corpuscles take a greenish stain and the granules are recognised as dark blue, sometimes almost black, points. They are equally well seen in eosin-methylene-blue-stained preparations; also when stained with Nicolle's thionin, and with Laveran's method. They are less noticeable after staining with gentian-violet or fuchsin.

These granulations are not seen in the living blood, nor when the films are merely stained with eosin. They cannot be mistaken for pigment, as the latter does not take a stain and has quite a different aspect. From Rubner's "Einführung zum Studium der Malaria Krankheiten," I learn that a similar appearance has been noticed in human blood taken from malaria patients. Rubner states that these granulations are identical with those which are known under the name of Plehn's plates, and that they are simply an alteration of the hæmoglobin of the red corpuscle and an indication of an anæmic state of the patient. These basic granulations may, in our case, be found in all the forms of distorted red corpuscles that are met with in the acute anæmia. They could be noticed in microcytes which were hardly more than 1 to $1\frac{1}{2}$ μ diameter, and then again in macrocytes whose diameter was from 10 to 12 μ . They were present in the plasma of the normoblasts or megaloblasts. In some cases the normal blood corpuscles would contain granules, and then it could be noticed that the corpuscles which contained them were in the majority of cases larger than those which were free from them. The granules were of different sizes; either the red corpuscles had the appearance of being powdered with Chinese ink, or dotted all over with points up to the size of a micrococcus of average dimensions.

Sometimes these points were all very big, and numbered from three to five per corpuscle; when smaller, up to thirty could be counted.

These different sizes of the granules gave me the impression that they originally start as very fine dust-like granulations, which gradually become confluent and form the larger granules. It is well to state here that the largest granules never reach the size of a pyroplasma (large form), are uniformly stained, and highly refractile, so that an experienced eye cannot make a mistake. There is still another change noticeable in the blood. When normal blood is stained with eosin and methylene-blue, we see that the red corpuscles are uniformly stained red; Ehrlich terms them orthochromatic. Sometimes, however, some of them do not stain a pure red, but rather a bluish-violet; these he calls polychromatic. Polychromatic erythrocytes were very frequent in the cases just described, showing all shades from violet-blue to a faint blue. Sometimes different shades of the bluish colour could be seen in one corpuscle. I have noticed this phenomenon in all forms of erythrocytes found in acute anæmia.

Very often the polychromatic cells contain most of the granules. Frequently it can be seen that a normoblast or megaloblast is polychromatic and contains the basophile granules. When such blood is examined in a natural state little else is noticed than that the red corpuscles are pale; the nucleus, however, is easily recognised, especially on the addition of acetic acid. In some of the nuclei of the normoblasts and megaloblasts a still more deeply-stained body can be observed, which is probably the nucleolus; and in some instances this nucleolus has fallen out, and the nucleus represents a ring. The nuclei were also found outside the red blood-discs, and were very often still connected with the lacerated plasma of the cell.

I did not come across granulations outside the red corpuscles.

I have already shown that these granulations and the other described pathological changes of the red corpuscles are also met with in cases of redwater which take a chronic turn or terminate in recovery. I have noticed since that Lignières has also seen the basic granulations in redwater blood taken from animals which recovered from the disease.

II. The Disease without Pathological Change of the Red Corpuscles.

This is decidedly the most common form, and its external clinical symptoms are in the majority of cases missing or indefinite, and a fatal termination is exceptional, and then mostly under the form of poverty when the cause is not looked for in the blood.

Ox 217. Weight 515 lbs., about 18 months old. Inoculated 21st December 1901 with 5 cc. virulent rinderpest blood and 60 cc. serum (No. 3). The animal went through the reaction of typical rinderpest. The fever, however, did not stop at the usual time, but continued irregularly, and a coincident loss of flesh suggested that it was suffering from some other disease besides modified rinderpest. On the 7th January 1902 the blood was examined and the trypanosoma found. Although the animal's temperature gradually went down, its condition did not improve; it went from bad to worse, and the animal was killed on 16th January. The blood was examined every

day from the 7th to the 16th January. The trypanosomata decreased in number, but basophile cells were never observed.

Post-mortem was made immediately after death. The blood was allowed to coagulate; it was of a watery character and a slightly brownish hue. The lungs were partly emphysematous. On the surface of the heart were subserous hæmorrhages, and the myocardium had white fibrous tissue in streaks. The gall-bladder was half full of thick bile. The spleen much enlarged, and its substance slightly pulpy. The kidneys were very pale. The fourth stomach contained big clots of coagulated blood; on the mucous membrane were hæmorrhagic ulcers covered with coagulated blood. The duodenum, ileum, and jejunum contained blackish fluid. The mucous membrane was swollen and spotted with ecchymoses. Peyer's patches were ulcerated and covered with necrotic membranes. The mucous membrane of the colon was also swollen, and showed disseminated hæmorrhages. Big clots of coagulated blood were found in the cæcum and rectum. The anus was lined with a layer of necrotic tissue. The lymphatic glands were enlarged, and some of them infiltrated with blood. The urine was yellow.

The severe lesions found in the intestines have to be attributed to rinderpest alone, which, owing to the presence of trypanosomata, very likely could not heal, although the typical time for rinderpest had elapsed.

Ox PL, two years old. This ox came from Pietersburg, and was infected on 12th April 1902 with 5 cc. virulent blood for the purpose of obtaining a supply of rinderpest blood to fortify the salted animals. A typical reaction followed, and when it was at its height, on 19th April, the animal was killed.

The *post-mortem* revealed nothing (beyond the characteristic symptoms of rinderpest of about four days duration) excepting a slightly jaundiced liver. The spleen was normal. There were some petechial spots to be seen under the endocardium of the left ventricle. The microscopical examination revealed the presence of trypanosomata.

Ox GG, four years old, came from Nooitgedacht, and was injected with 20 cc. blood of a goat infected with rinderpest. Eight days after this injection a reaction started, which turned out to be rinderpest. The animal was killed in collapse on the fifteenth day, and the *post-mortem* was made immediately. The symptoms of rinderpest were very pronounced. The spleen was slightly enlarged and hyperæmic. The liver jaundiced and also hyperæmic, also the kidneys. Trypanosomata were very numerous and accompanied by pyroplasma bigemini.

Calf 66. Texas calf, about twenty-eight months old, was inoculated with 100 cc. blood from an ox (61) whose blood contained the trypanosoma. On the fourth day after injection the trypanosomata appeared, and were present for thirteen days, their number being very considerable. High fever was constantly present. The animal became very weak, unable to stand, and finally died. On the day previous to death diarrhœa appeared. Though the animal fed up to the last day, emaciation set in and reduced it to a skeleton.

The *post-mortem* was made immediately after death. The carcase was in very poor condition, and general anæmia very marked. The lungs showed signs of hypostatic congestion. Heart, liver, and spleen

were normal. The bile was thick, and the intestinal tract in a state of catarrh, very pronounced in the colon.

Microscopic examination showed poikilocytosis, but no basic cells. The parasites had already disappeared from the blood for the last four days. A count of the red corpuscles during the presence of trypanosomata showed a reduction from 6,780,000 to 3,000,000.

Ox Carolina. Of this ox only microscopical specimens were at hand. The animal was in a herd at Carolina, which virulent redwater had already decimated. The specimens were sent to verify the diagnosis. No pyrosomata could be found, but trypanosomata were very numerous. The accompanying letter stated that the ox had been ill for a fortnight, and was near death when the blood was taken.

III. The Presence of the Trypanosoma does not Produce any Symptoms and has but little effect on the Animal.

We might include here the cases already described under PL and GG, where the trypanosoma was found in connection with rinderpest, and where on *post-mortem* but little traces of its presence could be noticed. Accordingly, the majority of the cases of trypanosoma infection ran an undetected course, because its presence in such cases did no harm.

Calf 47. This calf was infected with blood from Calf 57, in which trypanosomata were present; it received 50 cc. subcutaneously and 50 cc. intravenously. Trypanosomata appeared on the sixth day and were present during nine days. There was never anything amiss with the animal; its temperature varied from normal to 104° F., which latter temperature is not unusual in this country.

Calf 48, injected with 100 cc. blood of Calf 56 containing trypanosomata. Trypanosomata appeared on the fifth day, and were observed during nine days. The animal never showed symptoms of illness, although it was in pretty poor condition before the injection.

Ox 59. This ox was injected into the jugular with 100 cc. blood of Ox 56. On the fourteenth day the temperature rose to 106·8°, and trypanosomata appeared. They were only present for three days and disappeared again.

Experimental Results: Susceptibility of Different Breeds of Cattle.

I had the opportunity of testing cattle which came from different localities in South Africa, and from over-sea, with regard to their immunity against the trypanosoma. In speaking of susceptibility, however, I wish to make it clear that I am only guided by the appearance or non-appearance of trypanosomata in the blood after the injection. The severity of the results produced by the infection differs also in the different breeds, as will be alluded to in the course of this paper.

Altogether there were injected with blood thirty-eight animals, and the result proved that the blood contained trypanosomata, as they were apparent either directly or indirectly in the blood of some of the inoculated animals. Of the animals thus injected twenty-two showed trypanosomata, and sixteen none. These thirty-eight animals came from Africa, Madagascar, and America (Texas). Clinical observations could also be made on cattle from Argentine.

The different clinical observations relative to injection not performed for experimental purposes also came into consideration.

African cattle. Here are included cattle coming from the different parts of the Transvaal, from the Orange River Colony, and from Cape Colony. The following statistics are compiled from experiments on cattle which were purposely inoculated with the trypanosoma in varying quantities from 1 cc. up to 100 cc.

Of twenty-four infected African cattle, fourteen showed the trypanosoma and ten did not. Of these twenty-four cattle, fourteen came from the Transvaal, eight from the Orange River Colony, and two from Cape Colony.

Of these fourteen Transvaal animals, six could be infected and eight could not. Of the eight animals from the Orange River Colony, six became infected and two did not. The two Cape oxen which were inoculated both showed the trypanosoma. Of Madagascar cattle, two animals were injected, but neither took the trypanosoma. Of the twelve Texas cattle inoculated, eight animals showed trypanosoma.

From clinical observations the following may be added. The Argentine cattle, which apparently must have been infected by the blood used as virus in the simultaneous rinderpest inoculation, showed the trypanosoma in thirteen cattle out of sixteen, and probably if those which died in the earlier period after the infection had lived long enough they also would have shown them. The experience with Madagascar cattle is not sufficient to form a definite opinion as to their susceptibility. I have observed one Madagascar ox with trypanosoma. The Transvaal cattle are, in my opinion, less susceptible than any other breed.

The experimental results show 57.9 per cent. of susceptibility, but from clinical observations it has to be considerably lowered. I observed, about the time when the nature of the trypanosoma infection was discovered, that out of the many animals which were infected with one and the same sample of blood, only a few became infected and showed any disturbance in their temperature, or parasites in their blood. The same observation holds good to some extent in respect of cattle coming from the Cape, although more cases were clinically observed amongst them than amongst Transvaal cattle. About cattle from the Orange River Colony I only have the experimental results already quoted.

Morbidity.—The presence of the trypanosoma, as already shown, is not necessarily the cause of serious trouble to the animal in whose blood it is found. Its effect differs in different animals. We have also seen that the severest form of the disease is not necessarily followed by death, but in the majority of fatal cases there is a great destruction of red corpuscles described as basophile granulations. Taking the formation of these basophile granulations as an index of the degree of infection, then my observations lead to the conclusion that the Argentine cattle are the most susceptible (pyrosoma infection playing, without doubt, a considerable part in the observation). Then follow the cattle from the Orange River Colony, where, out of eight head, five showed these granulations distinctly. Next would come the Cape cattle, where I found the trypanosoma and basophile granulations in about 40 per cent. of all the cases, either together or

following each other. The Texas cattle, although susceptible in the proportion of 66 per cent., never showed any basic granulations. One death had to be attributed to the trypanosoma, where, however, only anæmia was present.

Susceptible Age.—I have observed the trypanosoma in cattle of all ages. The clinical observations were, with only one exception, made on animals from eighteen months to two years of age. In the inoculation experiments with Transvaal cattle, out of fourteen inoculated animals only six could be infected which were about one year old, and most of the eight animals which failed to take the infection were above two years old. The Orange River Colony cattle thus experimented upon were all under eighteen months of age. The Cape cattle in whose blood trypanosomata were seen were all full grown animals above four years of age. The Texas animals ranged from one to two years. The Argentine animals were heifers carrying their first calf. Out of four Argentine calves which were similarly treated, and therefore all exposed to the same infection, only one became infected. The Madagascar oxen were all full grown animals.

Mortality.—Out of a record of forty cases which can be considered as pure and simple trypanosoma infection, only five cases of death were registered, which would be equal to 12.5 per cent. The Argentine cattle are excluded from this list, because they were mostly cases of mixed infection with pyrosoma bigeminum.

Period of Incubation.—The duration of the incubation period depends greatly upon the number of trypanosomata which the injected blood contains. It averages between four and six days. An incubation period of three days only for doses from 50 to 100 cc. was observed in three animals after injection of 100 cc. of virulent blood. Inoculation into the jugular vein does not seem to have any shortening influence on this period. Of four animals which received 50 cc. respectively into the jugular vein and under the skin, three had an incubation period of five days, and one of six days. After the injection of 10 cc. blood with trypanosomata the incubation period was prolonged to ten days, and after the injection of 1 cc. a period of eighteen days was registered. In the above mentioned inoculations trypanosomata were present in the blood used as virus. The blood, however, may be infectious although no trypanosomata can be found on microscopic examination; but then we have to presume that they may nevertheless be there, though in very reduced numbers. In such a case the incubation period was found to be twenty days after the inoculation of 10 cc. blood.

Length of Time Trypanosomata are Present in the Blood.

This time may vary considerably, and it is, in my opinion, dependent upon the refractory condition the animal presents against the trypanosoma, as will be shown under "Immunity." The longest period in which the presence of the parasite was observed was thirteen days, the average was nine days, and the shortest period one day. The quantities of the injected trypanosomata has nothing to do with the duration of their presence in the blood. For instance, in the case of only one days duration 400 cc. of trypanosoma blood had been injected. In another case, where only 1 cc. blood was injected, the trypanosoma could be observed during three days.

Effect of Injection of the Trypanosoma.—The different effects which the trypanosomata have on the animal have already been dealt with. It remains, however, to show that the injection not only produces no result in refractory animals, as indicated in the statistics of susceptibility, but causes in some cases only a febrile reaction in which no parasites are seen at all, even in susceptible animals.

For example, in Calf 34, two years old, 50 cc. blood were injected into the jugular vein, and 100 cc. subcutaneously on 18th April 1902, from Ox PL., which contained trypanosomata. A reaction set in from about the eighth day, and kept on for seven days, during which no trypanosomata could be seen.

At the beginning of the reaction the number of red corpuscles was 5,710,000. This number dropped at the end of the reaction to 3,510,000.

Case 36, about five years old. This animal was injected on 2nd May 1902 with 20 cc. into the jugular and 15 cc. subcutaneous from Calf 12, which showed trypanosomata. A reaction began on the sixth day, and continued for five days, during which no trypanosomata were found. We have to assume that in such cases the number of trypanosomata is a very limited one, so that they escape observation. This may be illustrated by the following cases.

Cow 35, aged, received on 19th April 1902 400 cc. blood subcutaneously, and 100 cc. into the jugular from Ox PL. Reaction started about the fifth day, and during the reaction only one single trypanosoma could be seen.

Calf 26, ten months old, received into the jugular 100 cc. blood of Calf 56, whose blood contained trypanosomata. Only on two days during the period of reaction (six days) were trypanosomata seen.

Calf 56, one year old. Injected on the 26th August 1902, with 100 cc. blood of 36. A reaction began on the fifth day, when trypanosomata were seen; also on the following two days, but never again in the next twelve days during which the blood was examined.

Another observation made is the following: A reaction may set in after the inoculation and keep on for some days. During that period, however, no trypanosomata are seen, but they appear when the reaction is over, and may then be seen for some days.

Calf 77, injected on the 27th October 1902 with 100 cc. of blood from Calf 80 taken on the same day. On the seventh day reaction set in and lasted only four days, the temperature rising as high as 106.6° F. During this reaction no trypanosomata were seen. The examination was then discontinued for some days. On the fifth day after the reaction the blood was re-examined, when trypanosomata were found, and they were observed for the following eight days. As a rule when the trypanosomata appeared they could be observed daily, either in constant or in increasing numbers, but finally disappeared gradually. In some instances, however, it was noticed that they appeared and disappeared alternately, or, in other words, that they became in turns so reduced in number that they escaped observation.

Calf 69 was injected with 10 cc. of blood from Calf 77. Trypanosomata appeared on the eighth day. The next two days they were not observed, and then reappeared and remained for the following nine days.

Number of trypanosomata. It is difficult to give figures as to the

number of trypanosomata present in 1 cmm. They are never as numerous as, for instance, the trypanosoma Brucei in dogs, or the trypanosoma Lewisii in rats. I have counted as many as thirty in one field of a No. 6 Zeiss objective. An average of five may be considered a fair occurrence, although very often not more than one is found in a drop of blood. According to my experience, the number of parasites has not much influence on the severity of the disease or the lesions produced. It has already been demonstrated that in acute cases which led to death no trypanosomata were found on *post-mortem*, and they could only be traced by injection of blood into other animals. In cases where the basophile granulations appeared the number of trypanosomata did not exceed the average, and in some cases where the parasite were very numerous the animal seemed to be healthy.

Immunity. We have already seen that the injection of blood which contains trypanosomata is not necessarily followed by the appearance of trypanosomata in the blood of the injected animal, even not always by the reaction without trypanosomata.

This leads to the question whether such animals are immune, and also whether animals which pass through a trypanosoma infection are immune, or if they may be successfully infected a second or third time.

The following cases throw light on these points:—

Calf 9. (1) Injected on 16th December 1901 with 20 cc. blood of Ox 188. Reaction and trypanosoma made their appearance.

(2) It received on the 28th April 1902 again 100 cc. blood of Calf 12 subcutaneously. Reaction followed; no trypanosomata were observed, but basic cells.

(3) 18th July 1902. 850 cc. blood of Calf 57 (the blood contained trypanosomata) were injected subcutaneously and 100 cc. into the jugular. There was no reaction.

(4) 8th August 1902. 1500 cc. blood of Calf 54 with trypanosomata were injected. No reaction followed, and the examination of the blood remained negative.

Calf 12. (1) 19th April 1902. 100 cc. blood were injected subcutaneously and 50 cc. into the jugular of Ox PL. Reaction with trypanosomata followed.

(2) 200 cc. of blood of Calf 47 were injected subcutaneously and 100 cc. into the jugular. No reaction took place, and no trypanosomata were observed.

Calf 16. (1) 16th December 1901. 20 cc. blood from Ox 185, with basic cells but no trypanosomata, were injected. No reaction followed.

(2) 13th May 1902. 100 cc. blood were injected subcutaneously and 20 cc. intravenously from Calf 9, whose blood showed no trypanosomata, but basic cells. No reaction followed, and no trypanosomata were observed.

(3) 12th August 1902. 160 cc. blood of Calf 54 injected subcutaneously. This blood contained trypanosomata. No reaction followed, and no trypanosomata.

Calf 17. (1) 14th January 1901. 10 cc. of Ox 217, whose blood contained trypanosomata, were injected. No reaction was noticed.

(2) 21st April 1902. It received 100 cc. blood of Calf 12 subcutaneously. A reaction set in, but no trypanosomata were observed.

(3) 26th August 1902. 100 cc. of Calf 56 were injected into the jugular. The temperature rose on the fourth day to 105.6° F., but dropped again to normal the following day. No trypanosomata could be seen.

Calf 25. (1) 29th December 1901. 40 cc. of Ox D were injected. This blood contained no trypanosomata, but basic cells. A reaction followed, and trypanosomata were seen.

(2) 100 cc. blood with trypanosomata from Calf 12 were injected subcutaneously and 100 cc. blood into the jugular. An indistinct reaction was noticed, but no trypanosomata were seen.

(3) 8th August 1902. 100 cc. blood with trypanosomata from Calf 49 were injected. Only on the sixth day (for one day only) trypanosomata were seen, but no reaction took place.

Calf 26. (1) Received on the 16th December 1901 20 cc. of blood of Ox 188 into the jugular. This blood contained only basic cells. No reaction took place.

(2) 28th December 1901. 40 cc. of blood of Ox D were injected. Undecided reaction followed, but no trypanosomata.

(3) 26th August 1902. 100 cc. of blood of Calf 56 were injected, and were followed by a reaction and presence of trypanosomata during two days.

Calf 32. (1) Received on 3rd January 1902 35 cc. blood from Calf 25 with trypanosomata into the jugular. No reaction set in.

(2) 19th April 1902. 100 cc. of Ox PL were injected subcutaneously and 50 cc. intravenously. No reaction.

(3) 8th August 1902. 1000 cc. blood of Calf 49 were injected. Trypanosomata appeared, also basic cells and reaction.

(4) 16th September 1902. 800 cc. blood of Ox 61 were injected. Reaction set in, but no trypanosomata. The animal died, having become very anæmic.

Calf 33. (1) 19th April 1902. 100 cc. of blood of Ox PL were injected subcutaneously and 50 cc. into the jugular. A slight reaction followed, but no trypanosomata.

(2) 19th August 1902. 600 cc. of blood of Calf 56 were injected. The temperature rose on the second day to 104° F., the third day to 105°, and then dropped to normal. No trypanosomata could be seen.

Calf 33. (1) Received on the 19th April 1902 100 cc. blood of Ox PL subcutaneously and 50 cc. intravenously. Reaction followed, but no trypanosomata.

(2) 19th August 1902. After an injection of 600 cc. blood of Calf 56, there was again a rise of temperature but no trypanosomata.

Cow 35. (1) 19th April 1902. 400 cc. blood of Ox PL were injected subcutaneously, and 100 cc. into the jugular. Reaction followed, and one trypanosoma was seen.

(2) 16th July 1902. 3700 cc. blood of Calf 53 were given subcutaneously and 800 cc. into the jugular. No reaction.

(3) 8th August 1902. Received 1500 cc. blood of Calf 54. No reaction and no trypanosomata.

(4) 16th September 1902. 3600 cc. blood of Ox 61 were injected. No reaction and no trypanosomata were noticed. The animal died

on the 4th October 1902. General anæmia and poverty were the result of the different injections.

From these experiments we arrive at the conclusion that immunity exists to a considerable extent, and that it is brought about by infection with the trypanosoma. This immunity, however, seems to be dependent upon the number of trypanosomata injected; the greater the injection, the greater the chance of reaction or of the actual appearance of the trypanosoma.

DESCRIPTION OF PLATE II.

A. *Hippobosca* (*rufipes*?) natural size.

B. The same enlarged.

The following figures were drawn from the blood of Calf 4:—

- (a) Basophile red corpuscles.
- (b) Nucleated red corpuscles with basic granules.
- (c) Eosinophile white corpuscle.
- (d) Mononuclear white corpuscle.
- (e) Polynuclear white corpuscle.
- (f) Nucleated red corpuscle.
- (g) Trypanosoma.
- (h) Red corpuscle with pyroplasma bigeminum.

ON THE ETIOLOGY AND PATHOGENESIS OF OSSIFIC BLEMISHES.¹

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DURING recent years, and particularly since 1896, several important papers on the subject of ossific blemishes have been published, and have been the subject of long discussions at our professional societies, particularly at the Central Society of Veterinary Medicine. The time, therefore, seems opportune to attempt some generalisations based upon them, so that those persons who have not followed the discussions may understand the positions attained by the contending parties, and may be able to estimate the value of the accumulated reports.

On the 25th June 1896, M. Nocard, on behalf of M. Joly² of the Saumar School, presented to the Central Society a report entitled "An Investigation into the Subject of Splints" which was published by the *Revue Vétérinaire* of the same year.³ M. Joly showed that in addition to exostoses resulting from mechanical injuries, and therefore having no fixed position, and in addition to those ascribed to strain of the intermetacarpal ligaments, consequent on excessive vertical pressure on the heads of the small metacarpals, there occurred on the posterior margins of these bones what he termed "post metacarpal exostoses," produced by excessive tension on the carpal aponeurosis. This aponeurosis or sheath is relaxed when the foot is lifted, but becomes markedly tense when weight is placed on the limb. The to-and-fro movement at its lower point of insertion produces the kind of splint usually regarded as most troublesome,

¹ Translated from the "Revue Générale de Médecine Vétérinaire," No. 8, 15th April 1903.

² Etude sur la pathogénie des suros. Bulletin de la Société centrale de méd. vét. 1896, p. 487.

³ Etude sur suros. Revue vétérinaire. 1896, p. 589.





because, lying at the back of the limb, it is thought to interfere with the action of the tendons.

The first part of M. Joly's work seemed convincing, although M. Barrier thought that some importance must still be attached to the varieties of splint formerly recognised, viz., to intermetacarpal splint, to deep-seated metacarpal splint (*i.e.*, splint connected with suspensory ligament), to splint of the lateral ligament of the knee, and to splint of the lateral ligament of the fetlock, the occurrence of all of which is beyond dispute. It was therefore well to remember that while the exostoses termed a "splint" might occur in one of many positions, the common cause of all splints was strain or laceration of a ligament or aponeurosis at its point of insertion.

On the other hand, M. Joly, without formally denying the occurrence of intermetacarpal splints, remarked that they rarely appeared in an isolated form, and that he somewhat questioned the classic explanation, viz., that they were produced by strain of the interosseous ligament in consequence of descent of the small metacarpal. Movement in this direction would relax, rather than render tense the intermetacarpal ligament, the fibres of which run obliquely downwards and forwards. Intermetacarpal originate in the same way as post-metacarpal splints, viz., from hyperextension of the lower fibres of the carpal sheath, which produces an upward and backward strain.

Some objections were made to the latter view. M. Barrier said that the fibres of the intermetacarpal ligament crossed like the letter X, and that even slight movements of the small metacarpal, especially that of the inner side, might cause tearing of these fibres.

This first discussion focussed the question of the mode of formation of splints, which are the simplest form of bony blemish on account of their position on the shaft of the bone, and it led up to an interesting anatomical classification, by showing that all the points of insertion of ligaments and tendons into the cannon bone might become the seats of exostoses.

A general agreement seemed almost arrived at, when, in 1897, M. Joly¹ published a first series of "clinical studies," the greater part of which was devoted to spavin formation. This lesion is undoubtedly very difficult to explain, because the primary injury is complicated by the existence of the numerous articulations which together constitute the hock joint.

M. Joly showed that the prevalent idea (that spavin was a simple periostosis) was wrong. "In the Clinique of the Cavalry School," he said "M. Jacoulet has always taught that spavin is essentially a dry arthritis of the lower joints of the tarsus." This opinion had, in substance, been advanced by Cadiot and Almy in their *Traité de Thérapeutique Chirurgicale*. "Although spavin may occasionally be nothing more than a highly placed splint, yet in the majority of cases it invades the tarsal articulations. In such case it is sometimes superficial, due to strain of ligaments, and forms a sort of collar around the lower tarsal bone, thereby fixing them together; sometimes, and by far the most frequently, it is simply the outward sign of extensive dry arthritis, which results in ankylosis of the inflamed joints."

M. Joly sets aside the "ligamentous" origin of spavin, and sums up his conclusions as follows: In reality what is termed and what is

¹ *Études cliniques. Revue vétérinaire. 1897, pp. 522-574.*

treated as spavin results from a complex pathological process, the successive stages in which are as follows:

- "1. Dry arthritis of the lower tarsal articulations: spavin arthritis.
2. Anchylosis of the inflamed joints: spavin anchylosis.
3. Localised exostosis; in consequence of the anatomical formation of the hock, this appears on the inner surface of the lower part of the hock: spavin exostosis.
4. The disease attacks the lower tarsal articulations and invades the circumference of the tarso-metatarsal and upper tarsal joints: encircling spavin."

These phases may mutually encroach one on the other, but the articular lesion always appears first. The exostosis, in fact, is only an apparatus for preventing movement in a joint which is irremediably damaged. Even though the immediate causes of this arthritis must be sought in strains due to work, yet it must not be forgotten that defective conformation of the hock and of the limb exercises an important predisposing influence. And, in addition to external appreciable causes, there exists an individual predisposition resulting from the intimate structure of the bony tissue.

Heredity plays a very important part in the production of spavin, which often occurs even on very well-shaped hocks. The hereditary factor, according to M. Joly, is represented by a tendency to fusion of the individual bones forming the base of the hock; the "solipedisation" of the horse is still going on, and the pathological anchylosis of these joints is only an exaggeration of a normal process.

About the same time similar opinions on the origin of spavin were expressed by Eberlein¹ of the Veterinary High School of Berlin. This writer divides previously reported investigations into five classes:—

1. Those which refer spavin to injuries of ligaments or tendons.
2. Those which declare it to be a primary arthritis of the small articulations.
3. Those which regard it as a chronic deforming arthritis.
4. Those which think it originates in inflammation of the tendon sheath of the flexor metatarsi muscle.
5. Those which consider it to be a primary osteitis followed by arthritis.

One may, without disadvantage, combine the second and third groups and strike out the fourth, which no longer appears defensible, leaving only three principal heads of discussion, viz., those of—

1. The ligamentous origin.
2. The articular origin.
3. The osseous origin.

Eberlein supports the last view. In his opinion the primary change occurs in the depths of the bone, and consists in a rarefying osteitis; from this point it extends to the articulation, and the external lesion is the last in series.

As M. Barrier has very clearly shown, the fundamental point to discover is whether the morbid process develops excentrically, *i.e.*, whether it starts in the articular surfaces or interior of the bones and extends towards the periphery, or whether, on the contrary, its evolution is concentric, the process originating in the periphery of

¹ Der Spät der Pferde. "Monats. für prakt. Thierheil.," T. IX., 1897, p. 1.

the bones or articular surfaces and afterwards spreading towards the centre. After carefully examining a very large number of bony preparations, M. Barrier avows himself of the latter opinion, and declares that the lesions of spavin spread from the periphery of the articular surfaces or bones towards the centre, *i.e.*, concentrically, and that in almost all cases they originate in strains of ligaments. By arguments based on the anatomy and physiology of the parts, M. Barrier endeavours to prove that the row of bones beneath the astragalus is that which is exposed to the greatest strain during movement, and that of these the scaphoid and cuneiform magnum suffer the most. Further, he believes that any cause which tends to lessen the play of one of the anti-concussion mechanisms in a limb results in all the others being overloaded.

M. Barrier¹ also recalls—what has long been generally admitted—that youth is the time of life most favourable to the development of diseases of the apparatus of locomotion, because the bones and ligaments are then still imperfectly developed.

Whilst fully recognising the frequency and importance of the articular lesions, M. Barrier seeks to prove that the numerous anatomical specimens he has collected all show that the primary lesion is hyper-extension of the ligaments, especially of the short interosseous ligaments. This produces peripheral osteitis, which, by modifying the nutrition of the cartilage, leads to arthritic lesions. False or true ankylosis may follow, and in this way are produced the invisible spavins which are also due to ligamentous strains in the deep-seated portions of the hock.

Spavin ought, therefore, to be considered as at first an osteoarthritis, with a tendency to ankylosis and usually to deformity, commencing in the infero-internal portion of the hock, but sometimes extending from this point in all directions. The only doubtful point is whether the ligamentous injury is primary or secondary.

M. Barrier, like Eberlein, is opposed to the view that spavin is hereditary. He regards it as quite accidental; what *may be* hereditary is bad conformation of the hock.

This question of heredity is precisely what is at present most in dispute. M. Trasbot is strongly of opinion that spavin *is* hereditary, not only when it results from faulty conformation of the hock, *but even when it results from accident*; he only adds by way of modification, "I do not seek to discover if in this latter case the accident has been rendered possible by the defective quality of the parts."

Sanson, on the other hand, categorically denies the heredity of spavin, except in so far as it may be the result of defective conformation. Weber admits that animals may be predisposed to the disease by peculiarities of their osseous or ligamentous structure, and Menveux, while regarding spavin as a consequence of crushing strain of the articular surfaces, concedes that it may appear in young animals which have not performed any work, if their predecessors have been affected with the disease. In support of the hereditary theory, M. Magnin has cited cases from his personal experience, which led him to consider it dangerous to breed from animals suffering from spavin unless of known traumatic origin.

¹ Sur l'étiologie et la pathogénie de l'éparvin. "Bulletin de la Société centr. de méd. vét.," 1896, p. 339.

It was not long before propositions were advanced tending to rebut the views of M. Barrier. At the meeting on 13th October 1898 M. Joly¹ advanced fresh arguments, and stated clearly that he regarded the changes in the bone as constituting the primary lesion; the development of spavin is preceded by rarefaction of the bone, *i.e.*, by osteoporosis, which seldom remains confined to the joint first attacked, but affects other parts of the skeleton, as shown by the frequency of fractures in animals exhibiting many osseous blemishes.

On this argument is based the theory of osteitism. The most serious evidence in its favour is the frequency with which the spavin affects both hocks, a fact which can scarcely be explained except on the basis that this region is particularly weak. On examining one of these cases of ostitis after death, numerous osteophytes are found in widely separated regions, but all marking ligamentous insertions; they are accompanied by multiple lesions of arthritis and peri arthritis. A constitutional predisposition to osseous blemishes in general appears to exist, and this condition might very well be transmissible. The question of the heredity of some particular structure of bone is therefore closely related to that of the heredity of conformation. The collection of cases described by M. Joly bearing on this point deserves serious consideration. The general condition it presupposes would be a factor not only in determining hereditary peculiarities in individuals, but also in families. The Anglo-Norman seems particularly affected by this condition, as is the Anglo-Arab, and in general all crosses with the thoroughbred.

The repeated occurrence in series of this lesion, in one family, would bring about congenital fusion of the rows of bones usually attached, and, as a consequence, the simplification, from a mechanical point of view, of the structure of the hock.

A long discussion ensued,² but it is clear that from this moment no decisive argument was advanced on one side or the other, and that new facts must be sought.

About the same time M. Blanc of the Lyons Veterinary School, published the result of his anatomical investigations regarding sidebone.³ From microscopical examination he concluded that sidebone results from lateral extension of the third phalanx, the osseous tissue having a tendency to invade the lateral cartilage. Any cause capable of producing congestion of the last phalanx may assist this process of ossification. Sidebone rarely results from direct injury.

M. Mouilleron also supports the latter view: of 1993 cases of sidebone, only 259 appeared due to mechanical injury.

Udriski⁴ of Bucharest also published a note on ringbone. His conclusions are based on anatomical and pathological grounds. By a special method of investigation Udriski proved that the primary lesion most commonly affected the bony tissue. The change commenced in the compact portion of the bone, appearing first beneath the articular cartilage and next at a point some millimetres distant,

¹ Au sujet de l'éparvin. "Bulletin de la Société centr. de méd. vét.," 1898, p. 577.

² Sur la pathogénie de l'éparvin. "Bulletin de la Société centr. de méd. vét.," 1898, p. 685.

³ Etudes sur l'ossification du cartilage complémentaire de la troisième phalange chez le cheval. "Journal de méd. vét.," 1898, p. 193.

⁴ Die path. Anatomie der Krongelenkschale des Pferdes. "Monats. f. Thierheil," T. XI, 1900, p. 337.

beneath the periosteum at the points of attachment of ligaments. The deeper layers of the cartilage were destroyed; the ulceration which thus resulted was sometimes duplicated on the opposing articular surface, and, at a later stage, total ankylosis occurred. In all cases the ligaments underwent marked change, and, in some, false peripheral ankylosis followed in consequence of ossification of the structures uniting the bones. The stage of rarefying osteitis was followed by condensing osteitis of a reparative character. The process tended to recovery. In his summing up Udriski admits the possibility of a double origin for ringbone: the first a primary central osteitis; the second a peripheral osteitis affecting the ligaments.

Eberlein and his pupil Kärnbach¹ divide ringbones around the coronet joint into three distinct varieties, according to certain anatomical principles. In the first the lesion commences as a primary rarefying osteitis which leads to arthritis and periarticular hyperostosis. In the second the disease commences with osteitis of the subperiosteal layer of bone, leads to formation of periarticular exostoses, and at a later stage produces chronic arthritis of the joint. In the last the point of origin is the exterior of the bone, and the disease results from injuries to neighbouring parts. Ringbones therefore may originate either on the articular surface or in the subperiosteal layers of bone. The lesions exactly resemble those of spavin, but the process may after all be only a phenomenon in an evolutionary change ultimately destined to unite the several portions of the lever formed by the phalangeal bones.

In 1899, before Udriski and the German authors published their observations, M. Joly had advanced the opinion that ringbones were due to an osteo-arthritis occurring either, (1) without exostosis and without ankylosis; (2) with exostosis and without ankylosis; or, (3) with exostosis and ankylosis, the primary lesion being a general change in the bone, but always appearing first at the points of attachment of ligaments or tendons.

In animals suffering from such changes unexplained fractures of the pastern sometimes occur, in which after death the bony substance is found to be rarefied and affected with osteoporosis; the animals are in fact suffering from an osteitic diathesis. They show the plantar osteophytes described by Smith, navicular disease, subacute laminitis resulting from rarefying osteitis of the os pedis, and ringbones on the lateral surfaces of the os suffraginis, which Siedamgrotzky regarded as due to tearing away of the ligamentary structures which unite the lateral cartilages to this point.

This general condition, termed by the French "osteitis of fatigue," is the "occupational disease" of the horse; it produces such a condition of the bony structures that the slightest ligamentous strain may cause the production of an exostosis, and it extends to all parts of the bony skeleton, though affecting different parts with different degrees of intensity. Once acquired by an animal, it may be transmitted to the progeny, and bring about progressive degeneration. Its effects are particularly marked in the third phalanx, at which point it plays an important part in the development of chronic laminitis, and of those

¹ Zur path. Anatomie der Krongelenkschale des Pferdes. "Monats. f. Thierheil," T. XI., 1900, p. 516.

obscure lamenesses due to bone disease which affect race-horses, and which have recently been described by M. Cagny.¹

To assist in collecting material for the solution of this problem, the Central Society nominated it as a matter for discussion for 1902. M. Berton on the one side, and M. Vivien on the other, each contributed a voluminous essay; the former has recently been published.

M. Berton² frankly adopts the theory that ligamentous strain firstly produces exostosis around the joints, and secondly some form of osteoarthritis. He concludes with these words, "These pathogenic indications afford little foundation for the osteitic theory, which presupposes a condition in itself capable of causing the majority of bony blemishes, or at least such a predisposition that these same lesions may be produced by the most trifling accident in movement." On the other hand, M. Vivien, who was M. Joly's pupil and loyal colleague, entirely supports the ideas of his master. He says "The ostitis of fatigue originates in the depths of the bony tissue and rapidly extends. If in its course it encounters an interosseous ligament it attacks it, brings about its absorption, and induces the formation of a bony union. Should it, however, meet in its passage with an articulation, it destroys it, and causes ankylosis. It converts cartilage into bone, and if no tissue having intimate relations with bone, to which therefore it may extend, is to be found in its neighbourhood, it ends by causing the formation of a quantity of new tissue."

Like M. Joly, M. Vivien only regards the changes in bone which follow violent exertion as due to an exaggeration of that evolutionary process which has by successive stages reduced the manus of the soliped to a single finger. He does not regard ligamentous strains as playing any important part.

Quite recently I have attempted to explain several points not covered by the theories above mentioned.³ The nutrition of the bone appears to me to play the most important part in the predisposition to exostosis. This nutrition itself is dependent on the composition of the food; in some districts the horse's skeleton never becomes fully ossified. The insertions of the ligaments and aponeuroses into these unduly vascular tissues (which still exhibit some of the characteristics of foetal bone) are weak and liable to injury. Such defects of development are commonest in marshy countries with acid soils, deficient in phosphates, of which the Rochefort Marsh and the Lower Vendée region are typical. I know from personal experience that certain acid feeding materials may bring about general changes in the nutrition of bone, partly indicated by the frequency of fractures and tearing away of ligaments, and partly by the formation of bony growths. In 1900 I exhibited at the Central Society⁴ certain types of general metacarpal ostitis which almost always occurred symmetrically in the same subject, and appeared clinically as large growths of new bone enveloping the entire cannon bone. These periostitic growths were produced by the pull of the aponeurosis and ligaments. The articulation itself was seldom affected, but the entire thickness of the bone

¹ Les boiteries d'origine osseuse sur les chevaux de course. "Bulletin de la Société centr. de méd. vét.," 1900, p. 132.

² "Étiologie et pathogénie des tares osseuses du cheval," Vol. I., p. 148. Toulouse, 1902.

³ "Bulletin de la Société centr. de méd. vét.," 1903, p. 98.

⁴ Idem, 1901, p. 249.

was attacked. Very frequently the process ended in fracture from the most trifling cause.

In connection with this form of metacarpal ostitis, MM. Lefebvre and Thary described an osteo-arthritis of the knee, which I have also frequently noted, and which, starting in a synovial cavity, rapidly produces ankylosis of the lower chain of bones. There appears, therefore, to occur in the knee, as in the hock, two types of bony lesion, one localised in the periphery, the other in the joint. Regarding the part played in the production of bony blemishes by the food, many observations have been reported. I need only cite the work of Germain, of Theiler,¹ of Tapon,² of J. Dumas,³ and lastly a very suggestive paper by Prof. Marcone,⁴ of Naples, in which the author shows that the mysterious cases where tendons and ligaments are torn through without apparent cause depend on commencing general osteoporosis—in fact, latent osteomalacia without external signs, but with very marked microscopic lesions. In these subjects examination of the urine shows that the animals are losing phosphates; in fact, that their system is being drained of mineral salts.

Having thus recalled as impartially as possible the principal opinions emitted during the last few years, I may attempt a rapid synthesis.

We may begin by putting on one side all bony growths which result from direct injury or from suppuration in the neighbourhood, as well as those due to the action of specific microbes, mycotic parasites, or tumours. None of these are concerned with the point at issue.

(a) *The Importance of Individual Predisposition.*—Bony blemishes, though frequent in certain of the equidæ, are almost unknown amongst others. The ass rarely shows them, in spite of its being so often overworked. The mule has inherited from its father a large share of this immunity. Amongst horses, bony blemishes are particularly frequent in certain races; much rarer in others. The Arab and Breton horses are famed for the excellence of their bone. In all races youth constitutes an unquestionably predisposing cause. The bone has not then acquired its full solidity, and the ligaments are not so intimately attached to it.

Finally, *there undoubtedly exists a special susceptibility and an individual immunity.* Every practitioner has seen apparently worn-out horses which for years had performed extremely hard work without having developed the smallest bony blemish. Others again, though apparently well-shaped, begin to develop bony blemishes on the slightest exertion. Between these extreme types all varieties may be found. It seems logical to seek the cause of these variations in some difference in the intimate structure of the bone, on which again, without doubt, depends the weakness of the ligamentous insertions.

(b) *The Part Played by Mechanical Formation.*—It is remarkable that bony lesions are chiefly localised in the lower portions of the limb, below the knee and hock, and are almost unknown in the upper. This would appear to indicate that concussion is much less perfectly neutralised in the lower portions of the limb, the joints of which

¹ Osteomalacie. "Schweizer Archiv. f. Thierheil," 1895, p. 38.

² Des formes coronaires chez les jeunes chevaux. "Bulletin de la Société centr. de méd. vét.," 1894, p. 343.

³ Idem, 1896, p. 514.

⁴ "Osteomalacia del cavallo." Naples, 1901.

become more and more rigid as one nears the ground. Slips, twists, and repeated concussion, therefore, make their effects particularly felt at this point. In the majority of cases the determining cause is strain of ligaments; and, as in slipping the limb usually passes in an outward direction, the internal ligaments are those most commonly injured. We may therefore deduce the general rule that every insertion of a ligament, aponeurosis, or tendon, into one of the bones of the lower portion of the limb, is capable of producing a bony blemish should it be strained, and there exist as many varieties of exostosis as of points of insertion. The same principle extends to the periphery of the pedal bone, the podophyllous tissue in this case playing the same part as a ligament. There are very many cases where mechanical violence sufficiently accounts for the origin of exostosis.

(c) *The Part Played by Chronic Fatigue.*—Repeated concussion appears capable of bringing about changes in the bony substance in certain animals. Hence the origin of "ostitis of fatigue." Mechanical injury is probably not the only cause, but it is certain that the true substance of the bone is first attacked, the change afterwards extending to the articular surfaces, and then to the periosteum (as in certain varieties of spavin, osteo-arthritis of the knee, and ringbone).

(d) *The Part Played by Food.*—Nutrition of the bone depends on the food supplied; and that certain substances can diminish the resistance of the bone, while others can increase it, is shown by Wegner and Binz's experiments with phosphorus, which produces eburnation.

Growing horses fed with materials rich in phosphates, and particularly with grain, are likely to grow faster and have more resistant bone than those restricted entirely to grass. Foods grown on certain acid soils, poor in phosphates, cannot furnish the material necessary for the full development of bone, and some may even introduce into the body substances injurious to the bony tissue. Reul¹ has recently published in the *Annales de Bruxelles* an article of high importance regarding the part played in feeding by phosphates. Change of nourishment is often sufficient to check the course of this ostitic disease.

(e) *The Part Played by Heredity.*—Just as parents may transmit to their descendants defects of conformation, so they may transmit defects in the minute structures of the bony tissues. Animals with badly formed or defectively developed bone should therefore not be used for breeding. Blemishes resulting from accidents or mechanical violence are no more hereditary, however, than are strains or fractures. It is unfortunate that selection is confined to males; the female element is at least as important in determining the constitution of bone.

(f) *The Part Played by Evolution.*—Whilst quite admitting the possibility of further simplification of the skeleton in the horse tribe, I do not regard the pathological conditions above dealt with as a consequence of that evolution. Ankylosis of the scaphoid and cuneiform bones is no more a phenomenon of evolution than the ankylosis of the pastern mentioned in Udriski's work, or than that which in men accompanies the development of chronic rheumatism. Solutré's horses did not show any signs of bones united by new bony material simply because they had done no work.

To sum up, whilst agreeing with M. Joly regarding "ostitis of

¹ L'utilité des phosphates dans l'élevage des animaux domestiques. "Annales de méd. vét.," 1901, p. 410.

fatigue" and the ostitic diathesis which renders bones more susceptible to concussion and particularly to ligamentous strains, whilst recognising even the possibility that heredity plays some part in this constitutional condition, I do not regard heredity as always a factor, and refuse to consider the production of bony blemishes as anything more than a pathological condition independent of the evolution of the species.

A FURTHER CONTRIBUTION TO THE PATHOLOGY OF INTUSSUSCEPTIONS IN ANIMALS.

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IN the last number of the *Journal of Comparative Pathology* I gave a brief account of intestinal invaginations or intussusceptions found in man and animals. Of the latter I found records of over thirty cases. Through the kindness of Mr Henry Gray I have had my attention drawn to seven more, another has been found which had been previously overlooked, and I have had the good fortune to observe a case which occurred in the Zoological Societies' Gardens. Besides adding these records to my former list, one or two questions have been raised which have caused me to again contribute to this Journal. A brief resumé of the cases from the literature will be given first of all, followed by a more detailed account of the case observed myself, and then by any general remarks which seem advisable.

1. Henry Gray. *Veterinary Record*, 1902, XV., p. 131.

Intussusception of the small intestine into the large bowel, and protrusion of the small bowel outside the anus. This occurred in a chow-chow puppy of three months of age, who had diarrhoea for a few days, which was followed by prolapse from the anus. Operation was delayed for seven days, and the reduction followed by peritonitis following on a perforation of the bowel.

This intussusception obviously started in the ileum, which enteric invagination became prolapsed through the ileocæcal valve, so changing its name to ileocolic. Then the whole mass became invaginated into the colon, *i.e.*, a secondary colic intussusception, thus becoming a double intussusception of the ileocolic-colic variety.

2. Henry Gray. *Ibid.*, p. 191.

Female cat, seven and a half years of age, brought to him on account of a prolapse from the anus of twelve days' duration. Invagination reduced after a laparotomy. Bowel not gangrenous. The condition is described as "Eversion of the anterior or ileocæcal portion of the large intestine, and protrusion from the anus." The intussusception must therefore have been a colic one.

3. W. Pauer. *Ibid.*, p. 362.

Pony gelding, three years old, had been one week seedy and sluggish before the onset of subacute abdominal pain. *Post-mortem*—Two feet of ileum were invaginated into the cæcum. The intussusception was ileocolic, but is published as ileocæcal!

4. Captain Pickering. *Ibid.*, p. 305.

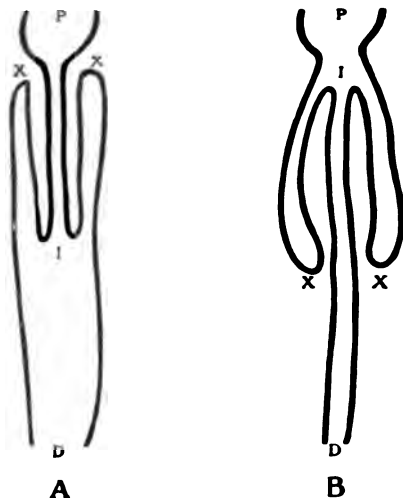
Gelding, attacked in the night with acute abdominal symptoms, pain followed by vomiting. *Post-mortem*—"Stomach distended with brownish

coloured fluid, and two feet from the pylorus the duodenum was intussuscepted back to the pyloric orifice, entirely blocking the outlet to the stomach, and carrying with it a portion of the mesentery." If this description reads aright, the above is an absolutely unique case of an ascending intussusception occurring during life. In view of the rarity of such an occurrence, it cannot but be felt that the above may mean that a descending invagination of the duodenum so pulled on the stomach that the pylorus became situated just above the ring of the inversion (A).

It would be a great benefit if Captain Pickering would settle this doubt. Fig. 1 (A) represents a rare but known condition; Fig. 1 (B) represents what I believe to be an absolutely unique condition.

5. W. M. Scott. *Ibid.*, p. 345.

Colt, aged six months. Muco-enteritis with "dysenteric" complications when nine weeks old. Acute symptoms ended in death in a little over twenty-four hours. *Post-mortem*—On undoing the invagination fifteen inches of gut were found involved. Unfortunately Mr Scott has omitted to say what portion of the gut was involved.



A represents a diagram of a descending intussusception in which the pylorus is at the ring of inversion (XX).

B represents a diagram of an ascending intussusception, in which the apex of the intussusception (I) is blocking the pylorus, and the ring of inversion is away from it.

P is the pylorus; X—X the ring of inversion; I the apex of the invaginated portion or intussusception; D the duodenum or small intestine beyond.

6. "Agricola." *Veterinarian*, LXVI., 1893, p. 145.

Cob. Death occurred within twenty-four hours of the onset of acute symptoms. *Post-mortem*—The apex of the cæcum was found inverted and so strangulated. Variety of intussusception therefore was colic.

7. Mr Henry Gray has attracted my attention to a case recorded by "Physalix" in the *Veterinary Record* of 22nd August 1903. Four pointers died after vaccination. In two an intussusception was found, and in a third Dr Arbel thought that an invagination had been spontaneously reduced. The morbid anatomy of the intussusception is not given.

8 "Petit." *Veterinary Journal*, 1903, IV. s. viii., p. 28. Extract from the *Recueil de Médecine Vétérinaire*, 15th April 1903.

Post-mortem on a dog that died of peritonitis which resulted from the chronic invagination of the cæcum into the colon. Variety of intussusception, colic.

The recent death of a gibbon, *Hylobates leuciscus*, at the Zoological Society's Gardens, led to the revelation at the autopsy of the existence of an intussusception or invagination of the gut. As the beast was one of considerable value, a careful inquiry was made into the history and condition of the animal with a view to the future recognition of such a state. A subsequent examination of the specimen removed, and at the Royal College of Surgeons, revealed the fact that the bowel was perfectly recoverable, and, had an operation been performed, the animal's life might have been saved and the Society spared the expense of the loss.

The gibbon had been in the possession of the Society for about a year. On the morning that she was taken ill she had been tremendously excited by the visits of a large number of soldiers. Just after her usual meal of fruit, bananas, apples, etc., she was seized apparently with great abdominal pains, followed by sickness and twisting about. The bowels were not open until "physic" had been given, and "blood and slime" were present in the first few evacuations. After this the blood in the motions disappeared. The sickness improved after three or four days, and then returned at intervals. Sometimes as long as four or five days would elapse between the attacks. All this time the bowels were open about twice a day, the motions being always loose and never formed. She never took her food well, got thinner and weaker, finally dying on the twenty-seventh day of the illness.

An examination of the specimen shows it to be an enteric intussusception, which had reached within six inches of the ileocæcal valve. The gut was not gangrenous, and had the invagination been reduced surgically there seemed to be no reason why the animal should not have lived.

SUMMARY.

Adding these seven cases to those published in the last number of this Journal, the results of the investigation of the veterinary cases may be summed up as follows:—

1. There are records of over forty cases.
2. Three occurred in cattle, thirteen in horses, twenty-five in dogs and cats, and one in an ape.
3. Of these cases there were four double intussusceptions, and about ten or twelve may well have been so.
4. Nine invaginations were of the small intestine alone—*i.e.*, were enteric. Twelve were ileocolic—*i.e.*, the invagination of the small bowel passed through the ileocæcal valve. Eight were colic, and solely involved the large bowel.
5. None arose at the ileocæcal valve, a point most important, as the pathologists have erroneously attributed the origin of the intussusceptions of men to this valve.
6. In dogs and cats the invagination seems chiefly to arise in the small gut. In horses, when very young, it starts in the small gut; when older, in the cæcum.

Intussusceptions are surgically recoverable accidents. The sooner that this is fully appreciated the better will be the treatment. This being the case, it is now time that emphasis be laid upon the diagnosis and recognition of the trouble, for then a long step will be taken

towards cure. Veterinary surgeons are far ahead of the surgeons of mankind, in that they select the cases which are worth operating on and have the others killed. The latter, unfortunately, cannot select their cases in this manner, and always operate in order to give the last chance to the subject, even though that is a very poor one. Experience shows that they are not often successful, or perhaps justified, in many instances. But the alternative of leaving the patient to die naturally explains their readiness to seize even at vain hopes. To operate is less inhuman than to leave to die. As a result, surgical procedures in such cases are far more often undertaken in human than veterinary cases.

It is of importance that the condition can be diagnosed. At present there still survives the discussion as to whether the diagnosis may be completed by the attitude and movements of the subject. The answer must obviously be "No." The behaviour of the animal is an index of the general condition which is caused by the local, rather than of the latter alone. In mankind the onset of any acute abdominal illness is the same—*i.e.*, abdominal pain, vomiting, shock, and collapse, etc. The condition has been christened by Sir Frederick Treves "peritonism." After the shock, etc., has passed off the condition becomes diagnosable, and remains so for an interval until the peritonitis produces obstruction by paralyzing the bowel, or the obstruction produces peritonitis either by perforation or penetration by organisms of the gut walls. In the first and third stage the condition is undiagnosable. The same will apply to the intussusceptions, "twisted guts," or ruptured stomachs of animals, with some slight modifications. For instance, shock and collapse is not nearly so frequently noticed in animals as in man. Another of the most interesting points in animals is their great tolerance of intussusceptions, and the consequent chronicity or subacuteness of the symptoms. In mankind, appendicitis and intestinal obstruction, other than by invaginations, form 62 per cent. of acute abdominal cases. Appendicitis is largely unknown in animals. Intussusceptions form 15 per cent., and perforations of the alimentary canal 11 per cent. Though of immense importance in man, the "acute abdomen" is not nearly so important in animals. But, from the point of view of comparative pathology, it would be very interesting if we could account for all the cases of so-called "twisted guts." As this is a subject of intense interest to me, I shall be glad if any veterinary surgeons would send me details or specimens with histories of such cases. And it is for this reason that my address is printed at the end of this paper.

A point upon which I wish to lay great stress is the tendency for veterinary surgery to swallow and incorporate with itself both the truths and untruths of human surgical science. I have pointed out in the *St. Thomas' Hospital Reports* for 1901 and the *Annals of Surgery* for 1903 that human surgeons base the diagnosis of the ileocæcal variety of intussusception on negative rather than positive evidence. And very naturally their pathology is wrong. Now this abuse is becoming veterinary as well. Mr Pauer calls his intussusception ileocæcal, but describes an ileocolic invagination, and numerous other examples might be quoted. By ileocæcal is meant that the invagination began at the valve which forms the leading part of the intussus-

ception or invaginated portion. This may be primary, or secondary to an intussusception of the small bowel, enteric, which pushes the valve in front of it. In no other sense should the term be used, and care should be taken that its use is based on positive and not negative evidence.

Finally, I would like again to draw attention to the agonal intussusceptions that occur during the "death agony," which are characterised by their multiplicity, being frequently of the ascending variety, easily reduced, accompanied by no signs of inflammation, etc. These are undescribed in animals.

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ON CERTAIN SEPTICÆMIAS AND SOME OTHER INFECTIONS OF YOUNG ANIMALS.¹

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FOR many years, in different parts of the world, raisers of stock have had to contend with losses of young animals which die a short time—a few days or weeks—after birth.

Deaths in young stock may be attributed to a variety of causes, but there is one cause now almost universally recognised, and which is included under the general name of septicæmia. That septicæmia, a very elastic term in this connection, is indicated on *post-mortem* examination most people who have had opportunities of a large number of *post-mortem* examinations will readily allow, but the question arises, what is the cause of the septicæmia, is there more than one cause, and how does infection occur? Then, again, do all animals that are infected die? or does the primary infection at times pass away, leaving the body deprived of its protection, open to the inroads of numerous saprophytes and parasites which otherwise would remain outside the body and harmless? Does the entrance of these organisms lead to serious lesions and perchance death, a secondary lesion, or may be lesions, developing as the result of their entrance into the body? I think it well, though it may appear to extend beyond the presumed scope of the paper as indicated by the original title, to consider these secondary lesions, as I hold that their value and importance is not inferior to the true cases of septicæmia which are more or less rapidly fatal.

But first, what is septicæmia? I consider septicæmia may be due to the presence and development of an organism in the body, it may be due to a toxin, or it may be due to both combined.

In a great number of septicæmias the causal agent may be more or less readily found in the blood, in other fluids or in organs, and inoculation of a minute quantity of material taken from such a case is capable of producing the same disease in suitable animals. To make my point clear, I consider anthrax in this sense a septicæmia.

In a second class we have an intoxication; the affected animal being poisoned by the toxin poured into the body. In this case

¹ Contributed to the proceedings of the National Veterinary Association, Sept. 1903.

inoculation of the fluids or juices of the body will not produce the original disease in animals of the same weight and size as the primarily affected animal, the amount of toxin in the fluid used for injection is too small, and will only produce any reaction in the smallest experimental animals where the dose of injection is relatively high when compared to the weight of the experimental animal. In this latter case there is no new formation of virulent material in the experimental animal such as in the first case, where the virulent material is capable of rapidly proliferating and giving rise to an immense new stock of virulent organisms. It is possible in this second form of septicæmia to produce an infection by injections of toxin produced by micro-organisms that have never been in the animal body.

If these two types of septicæmia are admitted, then the third is not improbable—a few micro-organisms producing a virulent toxin capable of producing death, an extension of the process to the body which occurs frequently enough in different, it may be isolated organs of the body.

It is perhaps to septicæmia of the first type—septic infection—that we must look as the chief form of septicæmia in young animals. Septic infection occurs through a wound either of skin or of mucous membrane, through any portal that will allow of the entrance of a micro-organism. But can infection occur through an uninjured membrane where there is no solution of continuity? This is not so certain, though many experiments, feeding experiments particularly, seem to show that ingestion of virulent material may set up disease through the alimentary tract. Swine fever, tubercle, anthrax, possibly black-quarter, may be cited. It is difficult to assert that there is no wound of such a membrane as that lining the alimentary tract, because it may be microscopic, merely a denudation of a few epithelial cells, and yet be sufficient to admit the organisms to the body. It is probable that where animal parasites are present in the gut the condition of the mucous membrane is rendered favourable to the entrance of organisms. Young animals, a few days old, however, are not commonly the hosts of intestinal animal parasites.

The skin when intact offers practically an insuperable barrier to the passage of organisms, though the rubbing in of a pure culture of the staphylococcus pyogenes aureus gave rise to a crop of abscesses upon the apparently intact skin of the experimenter. The application, if violent, may have produced a wound, but in this case the cocci are said to have been arrested in the hair follicles and glands of the skin. Even if this is the case they were still outside the body, but it is probable that the toxin associated with them may have killed the epithelial cells, allowed the entrance of the organisms, and as a consequence the formation of an abscess. In most cases, however, I think we may consider that our septicæmias are not due to entrance of organisms through an intact epidermis, but that in most, if not in all, there is a wound. In the newly born there is such a wound, the umbilicus, and the position of the wound is favourable to soiling—to infection. As a rule young animals are born where hygienic surroundings are altogether absent. Frequently enough they are born on a floor soiled by excretions, and their

very helplessness favours the infection by the vain attempts to rise made by the newly born. Until quite recently no attempts were made to cleanse the umbilical cord nor to seal the umbilical wound, and that infection does occur through the wound and cord may be readily demonstrated in cases of omphalo-phlebitis, the umbilical vein being distended with pus carrying an immense variety of micro-organisms. The presence of pus in this vein and no where else discoverable in the body shows it is the primary centre.

I think therefore we are justified in concluding that septic infection occurs through a wound; that the wound may be in the mucous membrane of the intestines, or in the skin, or on the superficies of the body; and that most probably the umbilical wound is the main port of entrance. In some cases the above remarks apparently do not apply, as in septic pleuro-pneumonia in calves, a disease said to be contagious, but we shall refer to this again.

Of recent years the different forms of septicæmia, those grouped in the class of septicæmia hæmorrhagica, have multiplied or possibly, owing to better technique and advance of science generally, observers have recognised maladies which previously were passed over, or included under other forms, and hence it is difficult in a paper such as this to mention all the types recognised by authors. I hope, however, to touch upon the most important and better established.

WHITE SCOUR IN CALVES.

A disease very prevalent in the south-western parts of Ireland and investigated in 1901 by Prof. Nocard and myself. The mortality from this disease was very serious, indeed many farmers were practically unable to rear any of their calves, and the yearly loss to the country became appalling. It has probably been prevalent for many years on isolated farms, but recently, in the decade 1890-1900, it became a veritable scourge. The disease manifests itself by diarrhœa, which usually appears on the second or third day following birth; rarely it makes its appearance on the first day, sometimes as late as the sixth; the farmer, if the calf shows no signs after the eighth day, thinks his calf will escape. The discharges from the bowels are thin, yellowish-white, frothy, and stinking. There is considerable tenesmus. The diarrhœa and straining soon render the animal so weak as to be unable to stand, and it lies constantly, soiled with its own dejections, moaning and grinding its teeth. Frequently there is a thin mucopurulent discharge from the nose which the calf makes no effort to clear away. The diarrhœa continuing becomes streaked with blood and bloody, the eyes sink in the head, the animal lies continually, groaning all the time. It refuses its food. It coughs, and sooner or later dies—a miserable object indeed. The temperature, at first high, may remain normal, or about normal for some little time, to fall and become markedly subnormal. I have observed a temperature as low as 92° F. just prior to death.

In some cases of acute white scour there is an intense arthritis of one or more joints. The joints particularly involved are the elbow, knee, stifle, and hock. The joint is hot and swollen and no weight is put upon the limb at all, the animal persistently lying, or, when up, standing with the limb flexed. Sometimes the limb between the joints

stifle, and hock, is also involved as well as the two joints. The muscles, intermuscular fascia, and fasciæ of the leg generally are infiltrated with a yellowish serum which gives rise to the swelling.

Post-mortem Examination.—The umbilical cord is thickened, soddened with a blood-stained fluid. The umbilical vessels are thrombosed, swollen, with the thrombus breaking down. The peritoneum joining the arteries is puce colour. Numerous petechiæ and ecchymoses found upon the peritoneum, especially on the serous covering of the stomachs. The liver is swollen but shows no gross lesions, microscopically there are minute areas of necrosis. The lymphatic glands are swollen, juicy, sometimes hæmorrhagic. The kidneys often congested, sometimes showing catarrhal nephritis. The urinary bladder contains a highly albuminous urine, and the mucous membrane is very vascular, swollen, and ecchymosed. In later cases there is a fibrinous peritonitis. The mucous membrane lining the first three compartments of the stomach is often diffusely hæmorrhagic; in the abomasum minute capillary punctiform hæmorrhages are common, the appearance on the washed mucous membrane is like a sprinkling of coffee grounds. The small intestines are congested, and the lymphoid tissue is infiltrated with blood and often breaking down. This condition is sometimes well seen in the large intestine, and especially when the fæces are blood-stained.

In the chest the pleura shows petechiæ and ecchymoses; the lungs areas of collapse, often small lesions of a hæmorrhagic pneumonia. The epicardium and the endocardium show beneath them minute hæmorrhagic extravasations. Similarly minute hæmorrhages into the substance of the valves may be found.

The lesions in acute cases are distinctly those of septicæmia. The sequelæ observed in animals that have recovered from an acute infection—white scour, are lung lesions, pyæmia, and necrosis.

The cause of the infection, as demonstrated by Nocard in Ireland, by Lesage and Delmer in France, and by Lignières in France and the Argentine, is an organism which belongs to the same family as that causing fowl cholera. This family is notorious for the septicæmic lesions produced, and is classified as that giving rise to septicæmia hæmorrhagica. Lignières has proposed the name *pasteurella* for the group, and names the diseases pasteurelloses. As showing the importance of these micro-organisms we may mention in passing that one or other of the family is the causal agent of fowl cholera, some forms of swine-fever, septicæmia of sheep, the disease of wild or semi-domesticated ruminants (wild seuche and rinderseuche), barbone des buffles, various forms of so-called influenza of the horse, and distemper in the dog. "The organism is a cocco-bacillus, non-motile, not taking Gram, polymorphous, not liquefying gelatine, not coagulating milk, which remains normal, does not grow on normal acid potato, does not produce indol in pancreatic bouillon, does not form acid, ærobic, but may be grown as an anærobe, produces an odour *sui generis*. No spores, no cilia. Of variable virulence; usually, however, it is high. When injected into the blood stream, the organisms show a predilection for serous and synovial membranes."¹

The organism of white scour, according to Lesage and Delmer,

¹ "Contribution à l'Etude et à la classification des Septicémies Hémorragiques," par J. Lignières, Buenos Aires, 1900.

may be found with many others in the intestinal discharges of suffering calves, and they say also in the bronchial mucus. Feeding experiments to healthy calves have failed to give the disease (Nocard, Lesage, and Delmer). Intravenous injection of a culture of the organism will kill a calf within thirty hours, and an adult cow which received 5 cc. of a culture died in twenty-six hours with most pronounced lesions of septicæmia. Application of a culture to the navel, either by rubbing on the culture or by tying on a pad of cotton wool soaked with culture, produced the disease (Moussu). Subcutaneous injections of the culture were not so successful. It appears, therefore, that entrance of the organism into the blood stream is essential to produce white scour, and that the port of entry in practice is the umbilical wound.

Thomassen has also described a septicæmia in calves associated with nephritis and urocystitis.¹ The disease affected calves in the neighbourhood of Utrecht, and proved to be very fatal. As the symptoms and lesions are different from those mentioned as observed in white scour, it may be as well to cull largely from the original paper and give them in some detail.

Symptoms.—The first symptoms are observed, not soon after birth, but towards the fifth to eighth day and even as late as four or five weeks. The animals have lost their spirits and lie constantly, either stretched out on the ground or with head turned back on the thoracic wall. Made to get up *they stretch themselves and bend downwards* (not arch upwards) *the back and loins*. (Particular stress is laid upon the symptom italicised.) The muzzle is dry and the respirations hurried. Pulse 100 to 150. The temperature is 104° F. and even higher. Sometimes there is a dry cough. Though the appetite is not good still the animal will take milk. Generally the droppings are normal in colour and consistence; they may be streaked with blood; diarrhœa has been noticed, but nothing approaching dysentery. The urine was often passed in small quantities; it was clouded, but red blood corpuscles were not at first suspected. Boiling with potash lye, however, gave a red colouration. It contained a large quantity of albumen, epithelial casts, and micro-organisms. The thoracic organs were sound. In some patients there were cerebral symptoms (somewhat similar symptoms were observed in calves suffering and dying from white scour. Toxæmia?).

The disease generally lasts some five or six days, and terminates fatally.

Lesions.—The lungs were found collapsed, the pleuræ were sound. No inflammation of the lungs. The blood in the heart was not coagulated, numerous ecchymoses upon endocardium and especially involving the valves. The bronchial lymphatic glands were swollen, softened, and showed capillary hæmorrhages. From the peritoneal cavity a clear fluid escaped on opening the abdomen, and the serous covering of the gut showed hæmorrhagic areas. The spleen was very much enlarged, even five or six times greater than normal; the capsule tense, violet, and glistening. The organ was soft, filled with blood.

There was, in all animals, a hæmorrhagic parenchymatous nephritis. The kidney on section was red, sometimes the redness being confined

¹ "Annales de l'Institut Pasteur," Tome onzième, 1897. Page 523.

to the medulla of the organ. The urinary bladder contained a highly albuminous urine with casts and epithelial cells and organisms. The mucous membrane was brownish-red in colour, uniformly or in streaks or patches. The ureters were also coloured.

The mucous membrane of the abomasum and small intestine showed ecchymoses, and the patches of lymphoid tissue were often swollen. The liver showed nothing pathognomonic. A slight meningitis with effusion was found in those cases where brain symptoms had been shown.

Thomassen shows that this septicæmia is different from that described by Jensen or Poels, and that it was not the septic pleuro-pneumonia of calves was evident, as the lungs and pleuræ were free from lesions.

It is also different from the malady we recognise as white scour, not only as to period of incidence of the disease, but also as to symptoms and lesions. Thomassen shows that this form described by him is due to an organism not taking Gram. It is allied to the colon bacillus (*B. coli communis*). It grows readily upon the usual media. Does not liquefy gelatine. It grows upon potato—which separates it from the class of organisms of which that causing white scour is a member; the culture, however, is an invisible one, which differentiates it from the colon bacillus and allies it with the organism causing typhoid fever of man. It is a motile organism. I have merely mentioned these particulars to show that Thomassen's organism is not that causing white scour. Further, it is more highly pathogenic than the usual coli organisms. Injected into calves, it produced death with all the symptoms and lesions in the experimental calves of the original disease. The organism is not pathogenic for the dog or horse.

Attempts at treatment failed. Carbolic acid in 2 per cent. solution was injected subcutaneously. Eucalyptol in olive oil was employed in like manner without result. Spirits of camphor per os, preparations of iodine injected into the veins, were all equally useless.

Another comparatively common septicæmia of calves is that known as septic pleuro-pneumonia, a disease first recognised in Holland, then in Belgium, Denmark, Prussia, France, Italy, and Russia. The organism found in animals dead or killed when suffering from the disease is similar in appearance, tinctorial behaviour, and in culture media to others of the septicæmic group. It affects calves, pigs, and kids. The symptoms of serious ill health are pronounced, and often the subject dies in a few hours after it is noticed to be ill. The young animals rapidly become weak, are unable to rise, refuse to suck or take food. In this malady the skeletal muscles are affected. The limbs become stiff, those in front are bowed, the hind limbs thrust far under the trunk. The animal moves with difficulty, and is evidently in great pain. Lung symptoms make their appearance, as noted by the respirations and cough, and by the soreness of the chest to palpation and manipulation generally. Auscultation shows dullness, râles, and tubular breathing. A foetid diarrhoea then sets in. Sweating, congestion of the visible membranes, frothing at the mouth, are also noticed. In young pigs the symptoms are allied to those of swine fever.

Lesions.—The lesions in the acute forms are found in the thoracic cavity. They are those of pleurisy—the effusion coagulating on exposure to the air, and ecchymoses are found. The lung is increased in size, hard and friable, and in appearance is likened to the lesions of contagious pleuro-pneumonia. The heart muscle has a parboiled appearance, it is paler than normal and friable. Lesions are found also in the abdominal cavity, but are less evident—petechiæ and ecchymoses upon serous and mucous membranes, and engorgement of the solid viscera with blood. The skeletal muscles also show small hæmorrhagic centres scattered through the muscles, blackish or a black violet tint. These centres may be a state of degeneration. The muscles may be infiltrated with serum. The causal organisms may be demonstrated in the lesions.

It is believed that this disease is spread by infection and by contagion. Poels thinks the organism is a facultative parasite, and that it is present in certain soils and upon forage, and that it obtains entrance into the body by the respiratory tract as well as by the alimentary route.

In the United States a form of septicæmia hæmorrhagica has been noticed which perhaps should be mentioned, because young animals die from it as well as older animals; indeed, neither age, nor sex, nor condition is proof against it. The evolution is rapid, death may occur in six hours, generally in twenty-four hours, rarely the animal lingers three or four days. The symptoms are interesting. There is loss of spirits, suppression of milk, loss of appetite, difficulty of movement, lameness and pain when moving. The pain is sometimes acute, the animals cry out and there is contraction of the muscles. If the animal lives more than twenty-four hours there is rapid loss of condition. The temperature, at first high, rapidly falls. Œdematous swellings appear in various parts of the limbs, and throat especially. The fæces, streaked with blood, and black, become watery and hæmorrhagic. Sometimes hæmaturia and blood-stained discharges are noticed. Lung lesions are not observed. [The symptoms are those of Wild and Rinderseuche, the clinical manifestations of which are little known.—Leclainche.] The lesions observed are those characteristic of septicæmia, blood extravasations of a varying degree being found upon the surface of membranes and in the interior of organs. The spleen is generally normal in appearance and consistence, as are the kidneys. In some cases the joint cavities contained a blood-stained synovia.

Jensen, who published the result of an experimental enquiry into the cause of diarrhœa in calves¹ in 1894, came to the conclusion that diarrhœa in calves was the result of a gastro-enteritis complicated with septicæmia, and that it was due to a pathogenic variety of a normal inhabitant of the gut closely related to the bacterium coli commune. His results were confirmed by others—Piana, Mazzanti and Vigezzi, Monti and Veratti, Piana and Galli-Valerio, and Romano, Italian workers. Nocard and Leclainche, however, are dubious as to the pathogenicity of the organism in question. It must be granted that the bacterium coli commune can very frequently be found in the tissues prior to natural death, the invasion occurring

¹ Ueber die Kalberruhr und deren Actiologie *Monatschrift für Thierheilkunde* IV.

when the animal is *in extremis*, and that its presence may be associated with the cause of the disease, though in reality it had nothing to do with it ; and, on the other hand, the effects of the invasion of the bacterium *coli commune* are so various that one need not be surprised that a variety of the organism may produce most fatal results. As regards Jensen's form of the disease it would perhaps be as well to keep an open mind, especially as his own results differ from those of workers who have recently investigated diarrhoea of calves.

Jensen's organism is motile, it produces gas in gelatine stab cultures, it coagulates milk, it grows upon potato, and the cultures have a foetid odour, characteristics which at once differentiate it from the organism of white scour.

The symptoms of the natural disease are like those of white scour, though in some cases the discharges from the rectum were greenish or green. Often in these cases of Jensen the materials first passed by the newly-born calf were liquid.

The lesions noted are as follows.¹ If the animal is opened immediately after death, the intestine is anæmic in appearance, but later it becomes dark red. The mucous membrane of the abomasum and small intestine is infiltrated, congested, studded with ecchymoses and desquamated patches. Peyer's patches are enlarged, the colon and rectum contain gas and a greyish viscid material mixed with little yellowish grains. The rectum is thick, red, inflamed, as is also the skin around the anus. Mesenteric glands are swollen and contain hæmorrhagic centres. The spleen and *urinary bladder* are not altered, (*italics mine*), the liver and kidneys are hyperæmic. The lungs are sound. Lesions of broncho-pneumonia sometimes found are due to foreign bodies. The myocardium is intact, the endocardium is ecchymosed." The condition of the urinary bladder here, should be compared with Thomassen's cases and with those seen in Ireland. The absence of lung lesions is significant. The lung lesions in the Irish calves were certainly not due to foreign bodies in the bronchi.

In this, as in all others, a knowledge of how the organism gets into the body of the calf is desirable. Jensen believes that the organism is always present in the alimentary canal of cows, and that it is expelled along with the fæces, soiling the parts with which they come in contact. The udder is contaminated when the cow lies down, and the calf takes in a dose of the organisms when it sucks the teat. Poels believes, on the other hand, that the infection is produced by the umbilical wound being soiled with the organism, which is a particularly virulent type of the colon bacterium. The umbilicus is infected during parturition or afterwards ; virulent organisms have been found in the vaginal mucus. Jensen obtained infection in seven calves, by feeding them on milk mixed with intestinal discharges of animals suffering from the disease. These calves were two days old ; older animals were not infected.

A particularly interesting experiment of Jensen's is worth mentioning. He gave newly born calves such substances as creolin, pyocyanine or trichloride of iodine, and saw them die within 48 hours with diarrhoea. The preparation given had injured the mucous membrane of the gut, and allowed intestinal organisms to cross the otherwise

¹ Nocard and Leclainche. "Les Maladies Microbiennes des Animaux," 3rd Edition, 1903.

efficient barrier and invade the body. These organisms recovered and grown in suitable media produced the disease when ingested by another calf.

It appears, then, that anything which will injure the natural defences of the body will allow of entrance of organisms which may produce death, and that these organisms are generally harmless when in the lumen of the bowel. This is probably the cause of death in many young animals—foals, calves, and lambs, which become the hosts of numerous animal parasites which wound the mucous membrane of the stomach and gut generally, and allow the access of micro-organisms which, under ordinary conditions remain outside the body in the alimentary canal. A similar explanation may be given for death as the result of retention of fæces or obstruction of the bowel. It is not so much absorption of the contents of the bowel—auto-intoxication—as it is injury to the bowel wall, which is rendered permeable to members of the intestinal flora. There is also considerable evidence in support of the contention that secondary lesions to a septicæmia from which the animals recover, are due to the entrance into the body of organisms which are normal inhabitants of the intestine, and which under ordinary circumstances thrive there without doing harm to the host. Indeed, it is probable that many members of the intestinal flora are absolutely essential to thorough and efficient digestion in the intestinal tube and to the normal development of the host, as witness Nuttall's experiments upon guinea-pigs fed upon bacteria-free food and kept in aseptic compartments, compared with others kept under ordinary conditions. Still, from the knowledge we have already gained as to the life history of organisms, we are driven to conclude that from some cause or condition or other, parasites or saprophytes which are ordinarily harmless may produce varieties which may give rise to serious lesions. The various coli-bacillary infections are examples of such. Perhaps better technique will demonstrate to us that what we now call varieties are quite distinct species.

Although it is somewhat beyond the scope of the original title of this paper, I think it well that mention should be made of several lesions that may be found in young animals, dying a short time—a few weeks, or may be two months or so—after birth. I mention these lesions because I am convinced that in many cases they arise as sequelæ to an antecedent septicæmic infection. Such secondary infections are the lung disease in calves, necrosis of the liver in calves and lambs, and pyæmia and pyæmic arthritis in calves, lambs, and foals.

LUNG DISEASE.

I have already mentioned that in calves dying from white scour, or in those killed in the later stages of the infection, lesions were found in the lungs, which varied from collapse to a hæmorrhagic pneumonia. The lesions are observed more particularly in the anterior lobes of the lungs. In some cases the lesions observed are those of bronchitis and catarrhal pneumonia, and it is to these last that I wish to direct attention. Calves that have apparently recovered from the "white scour" sooner or later begin to cough, and from the accelerated respirations, and from other clinical evidence, there is little doubt that the lungs are affected. The animals die in a month or six weeks

with consolidation of the anterior lobes of the lungs, the consolidation extending backwards, and formation of abscesses. I cannot call to mind a case where the posterior lobes have alone been affected, though there are cases where the posterior lobes to a greater or less extent are diseased with the anterior lobes. The lung on section is grey and solid, and studded with innumerable minute yellowish centres which are miliary abscesses. A pleurisy frequently is present, and adhesions, which may be so firm as to require severance with the knife, develop. The abscess formation in the lung continues, by the colliquation of the septa of adjacent centres which have become necrosed, and larger abscesses result. By an extension of the process the lobe may be replaced by an accumulation of abscesses, little if any of the lung tissue being left. At the periphery of the lesion a reaction occurs on the part of the still more or less normal lung, and attempts made to encapsule the lesion, and such is the condition observed in those animals which have apparently recovered from the lung affection. In some cases the lung involved becomes converted into a tough greyish mass, resembling in feel and in appearance morocco leather. The lung lesion is believed to result from an invasion of organisms found normally upon the respiratory mucous membrane, which during the septicæmic infection loses its power of preventing access of saprophytes into the tissues. The organisms found in the pus of the diseased centres are cocci, both staphylococci and streptococci, streptothrix, the bacillus of necrosis, the bacillus of caseous lymphadenitis. Nocard blamed particularly the last organism, and after he had injected the toxin formed by the organism of white scour into a calf he caused the calf to breathe an atmosphere charged with the organism of caseous lymphadenitis. In eight days the lesions had developed, and were similar to those observed in cases of the naturally acquired disease. But, though it is very probable that numerous cases of lung disease are due to entrance of the causal germs through the impaired bronchial mucous membrane, there are, I am convinced, other routes whereby the cause may gain access, and I think we cannot altogether exclude the umbilicus or other wound.

In this connection I may mention a number of cases I saw where there were lung lesions in calves that certainly never had white scour, calves that were collected from a very wide area, indeed I may say from the whole of Ireland. The calves in question were born about the end of March and the beginning of April. Some, to which we may particularly refer, were born in the most favourable surroundings, and the navel was treated in the approved fashion. There were never any symptoms of white scour. The calves thrived and were in admirable condition until the end of May. About the 26th May the males were castrated, and in about ten days from this date, on an average, the calves began to cough. Lung disease, clinically speaking, had put in appearance. I was now asked with another member of the profession to see the calves. We chose the worst for observation, and they were sent into the College. They had not long been in the College when the scrotum was found swollen, and in two cases a discharge appeared. The calves were then all carefully examined, and in every case a thickened or suppurating cord was found. In one instance the cord was excised and a piece of twine which had been used as a ligature was found in the centre. Two calves were killed

and found to have lung lesion; the same organism was found in the lung lesion as in the cord, and the organism was not the bacillus of caseous lymphadenitis.

I next made enquiries as to the relative numbers of the two sexes showing the lung lesions, and learnt that they were all males. The good health of the calves up to the time of castration, the appearance of the cough at or about the tenth day following the operation, the condition of the scrotum and cords, the presence of the same organism in both cord and lung, were suggestive as to the method of infection.

Another case which was brought to my notice by Mr R. Sargent, M.R.C.V.S., of Mullingar, is instructive. The patient was the last but one of a herd of twenty-seven calves; most of the others had perished. The calves were pedigree Polled Angus. The calf was sent into College living, and was kept six days for observation. It was in good condition, though coughing, and had all the clinical symptoms of lung disease. The temperature whilst in College never went above 39° C. (102.2° F.). The calf was killed by opening the carotids.

The *post-mortem* record is as follows: Umbilicus healed but thickened, no pus, umbilical vein sound and normal. Liver showed a few white specks under capsule, but otherwise normal. Spleen normal. Lymphatic glands throughout the body enlarged and juicy. Kidneys loosely attached (floating), fat dropsical, kidneys otherwise normal. In thorax both lungs involved nearly throughout, only sound part in posterior lobes. Anterior lobes showed large abscesses, and there was a pleurisy of some age. Adhesion to chest wall. The left anterior lobe united firmly to pericardium and filled with large abscesses. Smaller abscesses along sternal edge of lung. Where no abscesses save in posterior lobes, the lungs were broncho-pneumonic. In the mouth was an opening leading into a blind sac filled with caseous pus; the swelling produced by the sac had been noticed during life. Pus in the lungs contains the bacillus of necrosis and an organism taking Gram like the Preisz-Nocard organism (bacillus of caseous lymphadenitis). There was an indication of suppurative middle ear disease of left side. (Discharge from the ears had been noticed in this animal and others of the same herd.)

This calf had in the lungs typical lesions of the so-called lung disease, and yet white scour is unknown in the district. Clearly, then, in this case some other explanation than the primary infection by the organism of white scour must be offered to explain this invasion.

But there are other cases, and to my mind most instructive. Towards the end of March of the present year I received from Mr J. F. Healy, M.R.C.V.S., Midleton, Cork, livers from young lambs from one to two weeks old. An unusual number of lambs had died, and the livers were sent to me for opinion. I had little difficulty in recognising necrosis of liver, such as M'Fadyean described years ago as occurring in the livers of oxen. The areas were, however, larger than is usually the case, some wedge-shaped, others spherical, and embedded in the liver; one lesion was quite two inches in diameter. They were mostly firm, of a putty colour; others had a soft breaking-down centre. The bacillus of necrosis was found in apparently pure culture. I inoculated an emulsion of a necrotic area made with

bouillon into the auricular vein of a rabbit (1). This was on 20th March. On 26th March the rabbit was found dead in the morning. The thoracic organs were sound. There was a peritonitis, with a thick glairy albuminous fluid containing masses of coagulated fibrin. Necrosis of parietal peritoneum. Epiploon thick and infiltrated. Liver covered with exudate and united to diaphragm. Liver was necrotic, one lobe almost entirely so, and there were specks of necrosis through the organ, which was enlarged. The necrosis also had attacked the serous covering of the colon, and the wall of the bowel was necrosed from the serous membranes inwards towards lumen of bowel.

An emulsion was made from the liver of the above rabbit and injected into the auricular vein of another rabbit (6). It was killed twenty-two days later. The scapulo-humeral articulation was affected; a large abscess involving this joint, extending down the humerus to the elbow joint, contained in pure culture the organism of necrosis. The lesions were precisely those of joint evil. The humerus, head and shaft, was necrosed.

Another rabbit (13) received an emulsion in bouillon of the pus of last animal into the thigh. An abscess developed, and a necrotic area had developed in the liver; it contained only a few bacilli. A second rabbit (12) received into the auricular vein pus in bouillon obtained from rabbit with necrosis of the shoulder joint. The animal was killed four days later. The liver contained a lesion as large as a hazel nut with the bacillus of necrosis in pure culture. The urinary bladder was enormously distended with fat-containing urine—chyluria.

The pus from the abscess developing in the thigh of experimental rabbit 13 was inoculated after dilution in bouillon (1 cc. of dilution) into jugular vein of a red and white calf eight days old. The animal lost considerably in condition and the fæces were streaked with blood. The calf died on 14th May with intussusception of the cæcum, the mucous membrane of which was extensively necrosed. A large white rabbit was inoculated at the same time into the auricular vein with .5 cc. of same dilution as this calf. It was found dead on 6th May. The ear of injection became erysipeloid, but this passed off; the lymphatic glands behind the inferior maxilla became enlarged and purulent. On *post-mortem* an intense pericarditis, left-sided pleurisy, and pneumonia had developed. There was firm adhesion to the chest wall and to the diaphragm. There was a pleurisy on the right side, but not so severe. Small necrotic areas were found in the liver and on the visceral peritoneum. In this case the organism had attained an enormous length, and showed in a remarkable fashion the alternate presence and absence of coloration when stained.

I think these few experimental cases show the many-sided rôle the organism of necrosis may play, and that in these experimental animals we have epitomised the sequelæ observed in practice. It is interesting to observe that necrosis of the liver is merely a local manifestation in the liver of lesions occurring in the joints, in the connective tissues, in the serous membranes, and lungs. It is, further, to be observed that the lesion in the liver may extend through the diaphragm into the pleural cavities and involve the pleuræ and lungs; and I have had an opportunity, through the kindness of Mr Healy, of observing a necrotic centre extending through the diaphragm and

involving both liver and lung. The centre had broken down, and contained the bacillus of necrosis.

Similarly, through the courtesy of Mr John Holland, M.R.C.V.S., of Athy, I received the liver of a calf that with others had died from white scour. It was a typical case of disseminated necrosis, such as one sees in adult animals, and which is so well illustrated in M'Fadyean's paper on this disease published in 1891.

Lastly, whilst upon this question of necrosis, I should like to refer to the disease described by Damman, and in which Löffler isolated an organism like to that of necrosis.

Animals of from one to four weeks old are affected. The patient loses its spirits and refuses to suck. The temperature is high. There is abundant salivation and difficulty in deglutition. Opening the mouth is painful; the tongue is swollen. There is a cough, and a muco-purulent discharge from the nose. Greyish patches of a variable size, isolated or confluent, cover the mucous membrane of the gums, tongue, palate, and fauces, formed of a fibrinous exudate, easy to remove, and covering an inflamed mucous membrane. The false membranes increase rapidly in thickness, form a thick, rough grey layer adherent to the tumefied and infiltrated subjacent structures. The jaws, throat, and intermaxillary space, etc., are the site of a diffuse inflammatory swelling. Sometimes the nasal chambers are affected, and the lesions may extend to pharynx, larynx, and trachea. In some cases the feet are affected, the hoofs being lost. Death occurs in from four or five days to three weeks. Damman described the disease as diphtheria of calves; and Löffler, who investigated it when working at diphtheria in man, had no difficulty in showing that there was no connection whatsoever between the two infections.

The lesions of this "diphtheria" of calves are interesting. On the mucous membranes the exudations mentioned above are met with, the mucous membrane itself is progressively necrosed, and the necrosis may extend to the subjacent bones and cartilages. The mucous membrane of the intestine is infiltrated and vascular, and diphtheritic patches of different sizes are to be met with in the cæcum, floating colon, and rectum especially. The lung also frequently shows lesions; probably these are of a nature of a foreign-body pneumonia. Ecchymoses on the serous membranes may also be observed. The disease has been transmitted experimentally to the lamb and calf.

I have mentioned at some length the symptoms and lesions of "diphtheria" in calves because I am convinced that a similar, if not the same affection may be observed in the United Kingdom. During the investigations into the cause of white scour, etc., in Ireland, I had occasion to visit many farms in the province of Munster. On three farms, one particularly, I found a number of calves suffering from what I described as necrosis of the jaws. The calves were all young and were still fed on whole milk. On examination of the worst cases I found that abundance of saliva was escaping from the mouth and that the animal was smacking the lips in a remarkable fashion. Peculiar elongated swellings were noticed along the line of the grinding teeth, even before manipulation. On opening the mouth the mucous membrane covering the gums carrying the incisor teeth was found covered with a greyish granular deposit which on removal exposed an eroded bleeding mucous membrane; some of the incisor

teeth had fallen, others were so loose as to be easily removed. The necrosis extended from the labial aspect of the gum, between the teeth, to the lingual surface. The elongated swelling noticed along the line of the molar teeth was found to be due to an accumulation of deposit, grey, granular, and easily broken down, like to that upon the gums of the incisor teeth. The mucous membrane was necrosed, and necrosis extended from the buccal aspect of the gum between the grinders on to the hard palate. Several calves showed the same condition, and I secured two heads for further examination, which showed me that the organism of necrosis was there in abundance. A thorough search through the viscera failed to discover any other lesion save one minute area in a lung, the size of a pin's head, which unfortunately was lost. All the calves, however, had large navels, and a careful examination of all the calves showed that those with necrosis had suppurating navels; the worst mouth case had a navel with a cavity in it capable of accommodating a large hen's egg. On enquiry the farmer informed me that the disease was well known, and that the calves recovered even when the mouth lesions were advanced, and he showed great reluctance to allow me to kill any of his calves, and I had to purchase.

On another farm two animals died exhibiting the same lesions as noted above. At a third farm I found what I take to be the earliest phase in the necrotic process. It was whilst making a *post-mortem* of four calves said to have died from white scour. The lesion was a minute excoriation, in this particular case upon the bare skin of the muzzle. Surrounding the pit was an areola of intense congestion. It is probable that in acute cases of septicæmia there is an exanthematous eruption—which is generally lost sight of in our patients owing to their hairy covering—an eruption similar to that observed in septicæmia in swine. Infection may then occur by the organism of necrosis through the abraded surface. I recently purchased in Dublin, among other calves for this and other work upon which I am engaged, a roan calf. From my notes I read as follows: "The calf when purchased had a diseased navel which was suppurating, hard, cord like, passing up into the body. During the time he was in the observation house he showed red spots on the bare skin of the muzzle. One healed leaving a pitted depression. Others succeeded upon the muzzle, upon the internal wing and inferior commissure of the anterior nares. The size that of a three-penny-piece and smaller." This particular calf died on 17th May 1903, and was found to have a purulent umbilical vein, as thick as the wrist, extending from the abdominal wall into the liver. The pus in the vein contained a large number of different varieties of organisms, including a *cocco-bacillus* not taking Gram.

Although my cases are not on all fours with those described by Damman and others, still I think I may claim they have a family resemblance, and are worthy of mention as an infection of newly-born calves.

Of pyæmia I do not think I need say much. It is already well known that joint-evil in foals and calves is largely the result of umbilical infection—a purulent omphalo-phlebitis. Lesions are frequently disseminated through the body, involving, it may be, an organ or many organs simultaneously. The clinical manifestations

most common and most striking is the pyæmic arthritis, and from this fact the common name of the infection. Pyæmia, even without joint lesions, is not unknown as a sequel to white scour, pyæmic lesions being recognised in the kidneys, heart muscle, liver, etc. The causal agent in these cases is most often a staphylococcus, but I have also seen a small streptococcus producing the disease in calves.

PROPHYLAXIS.

From what has already been written it may be inferred that in my opinion prevention is to be attained by careful attention to the umbilicus. But no matter how careful one may be in the umbilical treatment, this must be supported by cleanliness in and about the houses inhabited by the young and their dams. Treatment of the umbilicus may save animals; there is no doubt that thorough hygienic surroundings will cut down the mortality; both should theoretically remove altogether the cause of death. This has been our experience in Ireland. Sealing up the navel even in bad surroundings has undoubtedly saved a number of lives, but when to this is added as scrupulous cleanliness as practicable then the loss has become almost negligible. Count Moore at a conference held in Limerick gave his own experience in Tipperary, that whilst he had lost 75 calves out of 115 in 1900, last year he had only 2 deaths out of about the same number of calves born. Another gentleman, the vice-chairman of the Kerry County Council, though he had lost 120 during the past six years, last year did not lose one. Others spoke to the same effect. I know also of a celebrated breeding stud of thoroughbred horses that yearly lost a number of foals from diarrhoea, joint-evil, etc., last year after the navel treatment only lost one foal out of 59 born, and the single death was due to another cause than umbilical infection. I have therefore nothing more to say than that antiseptic treatment of the navel as speedily as possible after birth, preservation of the wound in an aseptic condition, and thorough cleanliness, will save many young animals at present lost. I am also convinced that this treatment will in great part, if not entirely, ward off the serious secondary infections which occur, and which I have endeavoured, I am afraid very imperfectly, to indicate at any rate in part.

That there may be other routes by which infection is brought about I am prepared at present to admit, but I hope that the time is not far distant when we shall be able to block every port of entrance, every vulnerable point, and thus save the lives of many young animals which previously have been doomed to perish.

I desire to acknowledge my great indebtedness to Messrs J. F. Healy, Midleton, Co. Cork; John Holland, Athy; M. F. Lynch, Youghal; R. Malone, Wexford; R. Sargent, Mullingar; G. H. S. Jarret, Longford; A. Dobbyn, Waterford; G. Newsom, Killoughter, Ashford, Wicklow; A. J. Moffett, Ballinasloe, Members of the Royal College of Veterinary Surgeons, for their courtesy and kindness in supplying me with material for this work.

The following publications among others, home and foreign, were consulted :—

Les Maladies Microbiennes des Animaux (to which I owe much),

Nocard and Leclainche, 3rd Edition, 1903. *Contribution à l'étude de la diarrhœa des Jeunes Bovidés et de l'entéqué (Pasteurellose Bovine)* by J. Lignières, Buenos Aires, 1898. *Revue Générale de Médecine Vétérinaire*, Toulouse. *Fortschritte der Veterinär-Hygiene*. *Journal of Comparative Pathology and Therapeutics*. *The Veterinarian*. *The Veterinary Journal*. *The Veterinary Record*. *Annales de l'Institut Pasteur*. *Contribution à l'étude à la classification des Septicémies Hémmorragiques*, Par J. Lignières, Buenos Aires, 1900. *Bacterienkunde und Pathologische Mikroskopie*, von Dr Kitt, 4th edit., 1903. *Contribution à l'étude de la Maladie des moutons*, Lignières, 1898. *Mortality among calves in Munster*, Dublin, 1901.

SIX CASES OF CARCINOMA OF THE OX.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

CARCINOMATA are regarded by many pathologists as occurring but rarely in the bovine species. The experience of the writer, however, has led him to believe that such is not the case, and that neoplasms having a carcinomatous structure are frequently met with in the usual routine of a large abattoir. The term "frequently" is used advisedly, but the writer does not wish to be misunderstood; all he desires to convey is that cancerous growths are more prevalent in the bovine species than is usually admitted.

It is interesting to note that all the cases here recorded occurred in Irish cows. The majority of cows exported from Ireland to this country have attained an extreme age, and are frequently in an emaciated condition.

The writer does not intend to review the many theories that have been promulgated from time to time as to the etiology of this disease, and he is content merely to record the results of a macroscopical and microscopical examination of six of his cases. These reports are given somewhat in detail, as he considers them of value in showing the manner in which the disease was disseminated throughout the system of the affected animal.

Case I.—Squamous-Cell Carcinoma of the Orbit of a Cow.

The subject was an aged Irish cow in good condition.

This animal, although evidently suffering acute pain, did not show any disturbance of pulse, respiration, or temperature.

The eyelids in the vicinity of the nasal canthus of the left eye were destroyed and replaced by a fungoid growth, which was irregularly circular, and measured about 10 cm. in diameter. The interstices were filled with coagulated blood and a glairy muco-purulent material. It emitted an offensive and penetrating odour.

On dissection of the parts it appeared that the neoplasm had had its origin in the vicinity of the lachrymal sac. An arm had penetrated the lachrymal duct and entered the middle meatus of the nose. The greater bulk of the tumour, however, had invaded the orbital cavity, and in its progress had pressed the eyeball upwards and outwards. As a result, the normal contour of the eyeball had been destroyed. The cornea was thick and opaque. The eye, so far as

vision was concerned, was destroyed. The neoplasm had not involved the tissues of the eyeball, or its muscles, or the lachrymal gland.

The cut surface of the tissues of the neoplasm showed that they were greyish in colour, fibrous in texture, and interspersed with fine points and lines.

The left sub-maxillary lymphatic gland was larger and firmer than normal.

The microscopical examination of the tissues of this neoplasm revealed that it was a squamous-cell carcinoma. It was composed of a connective tissue stroma, which showed a severe leucocytic infiltration and numerous alveoli of epithelial cells. The outer or germinal layer was formed of columnar cells, whilst those of the central zone were more or less flattened, and showed very little keratisation.

Case II.—Squamous-Cell Carcinoma of the Rumen of a Cow.

The subject was an aged Irish cow in fair condition.

Primary Lesion.—The wall of the rumen, to the extent of some 50 cm. by 18 cm. over the region of and mainly involving the anterior pillar, was much thickened. A considerable area of the surface of the thickened portion had undergone ulceration. The edges were prominent and overhung the floor of the ulcer. The floor was deeply fissured, and in the interstices was a glairy fluid mixed with food particles. The affected tissue cut with a well defined edge. An average section of the pillar of the rumen measured 10 cm. by 7 cm. The mucous membrane contiguous to the ulcer was considerably thickened, and was composed of two delicate layers lying alternately at right angles to the surface of the rumen. On close examination the one layer was seen to be translucent and homogenous, while the other was denser, somewhat granular, and yellowish. The mucous membrane covering the growth was irregular in thickness, and possessed the same macroscopic appearance as that already described as pertaining to the same tissue in the vicinity of the ulcer. Beneath the altered mucous membrane was a layer of dense glistening fibrous tissue, from which gross bands passed to join the central zone. The interstices thus formed were filled either with a gelatinous substance, or, more frequently, with a muco-purulent material, which on pressure could be expelled in ribbon-like masses. The central zone was composed of a dense glistening fibrous tissue interspersed with small irregular masses of a granular substance varying in colour from yellowish-white to light brown. The areas blended with the surrounding tissue, and when pressed by the finger the indentation remained. A considerable portion of the mucous membrane, as well as of the underlying tissue, was dark red in colour, evidently the result of hæmorrhage.

Metastatic Lesions.—In the wall of the rumen in the vicinity of the ulcer were numerous round, hard nodules, which varied in size from a small pea to a hazel nut. Their line of demarcation was seen to be, on section, sharply defined. They were firm, grey, and translucent. From the periphery delicate wavy lines of a denser white material converged towards the centre, which in some instances was of a yellowish colour. The mucous membrane superincumbent to these nodules was thickened and intact.

On the serous membrane of the rumen, over an area corresponding to the involved part, were numerous nodules, varying in size from a millet seed to a hazel nut. These nodules were isolated or grouped. They were firmly adherent to the wall of the rumen. They were grey in colour and firm in consistency. The periphery was composed of a translucent homogenous matrix, which towards the centre gradually acquired a muco-purulent character.

In many of the gastric lymphatic glands the glandular tissue was to a great extent replaced by a firm, somewhat translucent substance of a greyish colour, but which had a tendency to become soft, granular, and yellowish. The softened material could on pressure between finger and thumb be expelled in the same manner as inspissated lymph from a lymph follicle.

The omentum was studded with nodules varying in size from a millet seed to a large pea. They were either isolated or grouped in large irregular masses, loosely attached to the omentum, firm in consistency, and cut with a well defined edge. On section the smallest nodules appeared homogenous, but the larger were more granular in appearance.

The serous membrane of the peritoneal cavity on the left side and the peritoneal surface of the diaphragm on the right were studded with nodules varying in size from a millet seed to a horse bean. They were loosely attached to the peritoneum by bands of fibrous tissue. The larger were papillated on the surface. They were firm in consistency and greyish in colour. The cut surface was somewhat translucent and homogenous in appearance.

Microscopical Examination.—The stroma was in considerable amount. The epithelial cells penetrated the adjacent tissue. These at first were in delicate layers lying in the lumen of a lymph capillary, but ultimately increased in number. The cells forming the germinal layer of the alveoli were columnar, and possessed a large nucleus, in which in many instances were one or more nucleoli. There was a tendency to keratisation, typical cell nests being formed.

Case III.—Squamous-Cell Carcinoma of the Rumen of a Cow.

The subject was an aged Irish cow in poor condition.

Primary Lesion.—In the region of, and mainly involving, the anterior pillar of the rumen, at the point where its two branches are given off, was a fungoid growth measuring roughly 29 cm. by 18 cm. It had somewhat the appearance of an exaggerated cauliflower, and it was rudely crescentic in shape. The mucous membrane of the two ends was undergoing ulceration. It was firm in consistency. The cut surface was greyish in colour, interspersed with small white or yellow points and fine lines. These were softer in consistency, and could on pressure be expelled.

Metastatic Lesions.—Several of the gastric lymphatic glands in the vicinity of the primary lesion, and of the oesophageal chain, were larger than normal. The capsule of the lymphatic glands was tense and glistening. Their tissues were greyish in colour, somewhat fibrous in appearance, and firm in consistency.

Microscopical Examination.—The stroma was in considerable amount, and there were areas of dense cellular infiltration. The cells

forming the germinal layer of the alveoli were columnar in shape, whilst those of the central zones were more or less flattened, and showed a marked tendency to keratisation.

Case IV.—Squamous-Cell Carcinoma of the Rumen of a Cow.

The subject was an aged Irish cow in fair condition.

Primary Lesion.—In the vicinity of, and mainly involving, the anterior pillar of the rumen was an irregularly circular, fungoid, ulcer-like growth about 23 cm. in diameter. The edges were sharply defined, prominent, and overhung the floor of the ulcer, which was fissured, the

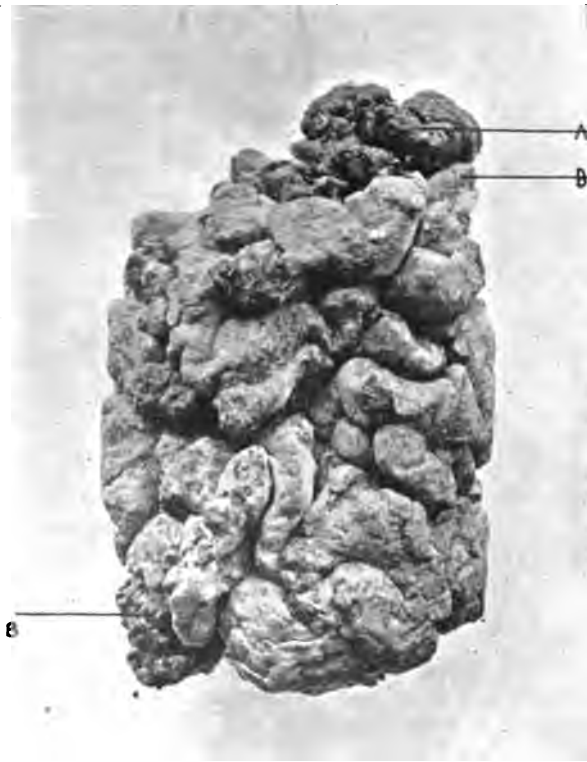


FIG. 1.

Primary lesion, Case III.

A, Pillar of rumen; B, B, Ulcerated areas.

intervening tissue having a fungoid appearance. In the interstices was a glairy fluid mixed with food particles. On section the neoplasm was seen to be composed of a thin superficial layer, yellow in colour and granular in appearance and in consistency, and a deeper and thicker one of fibrous tissue in which small granular areas were interspersed.

Metastatic Lesions.—Numerous firm round nodules, varying in size from a millet seed to a large pea, were present in the wall of the rumen

colour of the cut surface was a dirty yellowish-green, interspersed with areas of green and red, the latter being due to extravasations. The tissues at one side were laminated, and had the appearance of becoming organised.

In the muscular wall of the heart were a number of secondary lesions varying in size from a pea to a hazel nut. These were roundish in shape, with a well defined line of demarcation, which was irregularly crenated. The cut surface of these lesions showed that they were grey in colour and homogeneous in appearance, but there was a tendency to the deposition of a yellow granular material, either as slender lines or as spots. Numerous delicate blood vessels ramified through the tissues. In some instances these nodules protruded into the lumen of the auricles or ventricles as fungoid growths.

The right kidney weighed 900 grammes and the left 675 grammes. The contour of both kidneys was distorted owing to the presence of nodules in their substance. These showed on the surface as rotund projections varying in size from a millet seed to an orange. Their outline was in some sharply defined, whilst in others it was indefinite. They were, as a rule, yellowish in colour, but several of the smaller had a reddish tinge. In consistency they were firmer than the normal kidney tissue. On section numerous nodules were found situated in the cortical substance of both kidneys. These nodules were roundish in shape, yellowish in colour, and varied from a rape seed to an orange in size. The substance of these nodules was intersected by bands of a translucent grey tissue, which seemed to radiate from a central point. The tissues forming the centre of the larger nodules had, as a rule, undergone degenerative changes, and had acquired the appearance and consistency of pus.

In the loose connective tissue adjacent to both kidneys were a number of nodules varying in size from a millet seed to a hazel nut. These were more numerous in the vicinity of the larger lesions of the kidneys. They varied in shape, and their surface was, as a rule, uneven. They were of a yellowish colour. On close examination a number of minute specks and lines were discernible. Their cut surface was similar in appearance, and showed a few delicate wavy blood vessels.

The bronchial, the pericardial, the mediastinal, the prepectoral, the whole chain of supra-sternals on the left side, the right brachial, and the gastric lymphatic glands were involved. In the more advanced stages the glands were enlarged and nodular. The nodules were roundish in shape and varied in size. Their line of demarcation was sharply defined. The cut surface became slightly rounded. It was greyish in colour, interspersed with delicate yellowish lines and specks. In consistency the nodules varied from liver tissue to pus, the latter being those showing the deeper shades of yellow. Numerous delicate blood vessels ramified through their tissues.

Microscopical Examination.—The stroma was scanty in amount and well supplied with blood vessels. The outer layer of cells of the alveoli were more or less cubical, whilst those of the central zones were irregular and flattened. The nucleus was large, and contained in many instances one or more nucleoli. The cells showed little tendency to keratisation. The cellular elements of a number of alveoli had in a greater or less degree undergone fatty degenerative

changes. These changes, as a rule, commenced in the cells of the central zones.

Case VI.—Spheroidal-Cell Carcinoma of a Cow.

The subject was an aged Irish cow in poor condition. The *post-mortem* examination revealed the presence of a considerable number of nodules loosely attached to the peritoneum, to the omentum, and to the surface of both lungs, in the walls of the heart (these in some cases projected into the auricles and ventricles as fungoid growths), and in several of the muscles of the abdominal wall. These nodules varied in size, attaining the dimensions of a man's fist, and in shape



FIG. 2.

Heart of Case VI. cut open to show secondary lesions.

resembled an artichoke. Their surface was uneven and papillated. They varied in consistency, but the majority were somewhat soft, whilst their predominating colour was greyish-white, interspersed with reddish and yellowish areas. The cut-surface of these nodules showed a somewhat translucent matrix, intersected with white fibrous bands, which tended to converge from the periphery to the centre. In places the tissues had undergone degenerative changes, and cavities had resulted.

Microscopical Examination.—The stroma was, in areas, densely infiltrated with round cells. It was looser in texture in some nodules than in others. The alveoli were composed of large cells, which

varied in size, the majority being angular in shape. The nucleus was large, and frequently showed one or more nucleoli. In many of the alveoli were patches the cellular elements of which had undergone colloid degeneration.

DESCRIPTION OF PLATE III.

The six figures reproduce microphotographs of sections from the six cases described above. In each case the magnification was 100 diameters.

FIG. 1. Section of growth in orbit, Case I. Hæmatoxylin and eosin.

FIG. 2. Section of secondary lesion in the vicinity of the primary neoplasm, Case II. Heidenhain's method.

FIG. 3. Section of primary lesion, Case III. Van Gieson's method.

FIG. 4. Section of secondary lesion in liver, Case IV. Van Gieson's method.

FIG. 5. Section of secondary lesion in one of the gastric lymphatic glands, Case V. Hæmatoxylin and eosin.

FIG. 6. Section of nodule situated in one of the abdominal muscles, Case VI. Hæmatoxylin and eosin.

TUBERCULOSIS OF THE PENIS OF A BULL.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

AN interesting case of tuberculosis of the penis recently came under my notice.

The subject was a well-nourished three-year-old polled bull.

I am informed that during life it was observed that the penis remained protruded, and that there was a discharge from a growth situated in the left inguinal region.

The result of the *post-mortem* examination would, I think, be best stated as a review, commencing with what appeared to be the primary lesion. This was a nodule, situated on the inferior surface of the glans penis, about 1 cm. anterior to the attachment of the prepuce. It was rotund, soft, and measured 1.5 cm. in diameter. The epithelium covering it was intact. On section it was found to contain yellow caseated matter. In its vicinity were a number of small nodules, situated in the sub-mucous tissue. In close proximity, but posterior to the former, was another nodule, somewhat similar in size and having the same macroscopic characters. From these lesions organisms had extended to the loose areolar tissue surrounding the penis immediately posterior to the attachment of the prepuce. The penis had attained at this part a diameter of 17 cm. The growth measured 6.5 cm. in extreme length. It was mainly located on the left side, but it had extended and encircled the penis to the extent of about two-thirds of its circumference. It projected forward, forming an irregularly nodular but imperfect ring round the posterior end of the glans. It attained a thickness of 2.7 cm. at its most prominent part. The tissues of the growth were firm and elastic. The cut surface was intersected by bands of fibrous tissue, more or less apparent, into lobules. These varied in size from a millet seed to a hazel nut, and in shape were, as a rule, either round, oblong, or triangular. The tissues were somewhat translucent and homogeneous



Fig. 1.

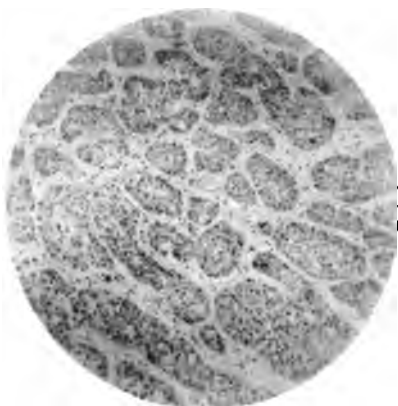


Fig. 2.

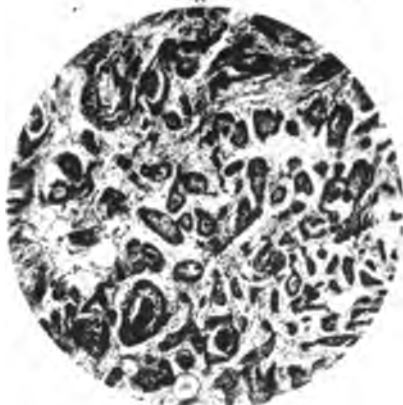


Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.



in appearance, and yellowish-grey in colour, interspersed with numerous caseated centres.

The underlying tissue of the corpus cavernosum did not appear to be involved.

The papillæ of the glans and, in a lesser degree, of the prepuce were prominent, and imparted an embossed appearance to these parts. The majority were of a reddish tint, and varied from scarlet to claret. The condition of the papillæ was probably caused through the internal and external irritation resulting from the constant protrusion of the penis during life.

Bacilli had been carried by the lymph stream from the tubercular lesions of the penis to the superficial inguinal and internal iliac groups of lymphatic glands, and had there been arrested. These glands measured in centimetres as follows: Superficial inguinal—left, 17·8 by 12 by 5; right, 12·5 by 10 by 3. Internal iliac—left, 9 by 9 by 5; right, 9·5 by 6·3 by 3.

On the left side several of the most posterior of the lumbar chain were also affected.

The tissue in the vicinity of the left superficial inguinal lymphatic glands had been invaded by tubercle bacilli, resulting in the formation of innumerable nodules. These, with the gland, etc., formed a mass which roughly measured 30 cm. by 19 cm. by 12 cm. An exploratory incision had been made into the growth some time previous, and the instrument employed had penetrated to the extent of 6 cm. into the substance of the anterior extremity of the gland. This wound had resulted in the formation of a fistula, the lumen of which contained a yellow purulent material. The inner surface of the walls, which were formed by thick fibrous tissue arranged in concentric rings, was dark green in colour, and emitted an offensive odour. Nodules had formed along the course of the fistula. The majority of the tubercular foci situated in the glandular tissue in the vicinity of the wound had undergone calcareous changes.

It is of interest to note that only lesions thus situated were found to have undergone calcification.

In addition to the foregoing, a few tubercular nodules were present in the substance of both lungs, as well as in the bronchial, mediastinal, and right and left prepectoral groups of lymphatic glands. These lesions, in all probability, were the result of the inhalation of tubercle bacilli.

On the peritoneal surface of the diaphragm was a thick tubercular deposit. Its superficial area measured 30 cm. in length and 19 cm. in breadth. Secondary lesions from this were present on the pleural surface of the diaphragm, and in the right xiphoid and the most posterior of the right chain of supra-sternal lymphatic glands.

The microscopical examination of smear preparations from the nodular mass on the penis, stained by the Ziehl-Neelson method, revealed the presence of tubercle bacilli in great numbers, whilst sections of one of the internal iliac lymphatic glands showed the usual characters of a tubercular lesion.

BURSITIS OF THE SUBTENDINOUS BURSÆ IN CONNECTION WITH THE GRACILIS, SARTORIUS, AND SEMITENDINOSUS.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

The subject of this note was a well nourished, aged, cross-bred cow. On the inner surface of each thigh was a growth. The one on the left side was the smaller of the two. It was situated over the inner surface of the tibia. It was heart-like in shape, but somewhat flattened through pressure against the bone. The superior margin, which corresponded to the base of the heart, was rounded, and was separated from the superior articular surface of the tibia by a distance of 4·5 cm. The growth measured 14·7 cm. in extreme length and 10 cm. in extreme breadth. It was covered by the fascia of the thigh, through which its surface appeared opaque, interspersed with a few indefinite yellow areas. The surface was uneven, the inequalities varying in size, and in consistence from dough to caoutchouc. On removing the fascia the tendons of the gracilis and sartorius were seen to pass over the outer surface of the upper portion of the growth.

The growth, on dissection, was found to be composed of three lobes, which were intimately bound together by fibrous tissue. A few blood-vessels were present in the interlobular connective tissue. The largest lobe occupied the superior position. It was somewhat round in shape and measured 9 cm. by 6 cm. by 2 cm. The medium-sized lobe formed the lower portion of the growth. It was triangular in shape, and measured 9 cm. by 7 cm. by 1·7 cm. The smallest lobe lay between the superior and inferior lobes at the anterior margin. It, in some degree, resembled in shape the navicular bone of the horse, and measured 5·3 cm. by 2·5 cm. by 1·6 cm.

The tendon of the semitendinosus, which passes forward to become inserted into the crest of the tibia, passed between the superior and inferior lobes and under the latter. Many strands of this tendon were inserted into the surface of the inferior lobe.

Each lobe was seen, on section, to have a cystic formation. Their walls were tough and leathery. The cut surface showed that each wall was divisible into two layers, but the line of separation was not well defined. The outer layer was grey, opaque, and somewhat laminated; the inner was amber-colour, transparent, and homogeneous. The wall varied in thickness from ·2 cm. to ·8 cm. The contents of these cavities were composed of a whitish-yellow granular material, similar in appearance to the overdone yolk of an egg, in which were embedded numerous hard, round, yellow bodies, varying in size, and which attained to the dimensions of a millet seed. On the addition of hydrochloric acid to these bodies an active ebullition of gas bubbles took place. These lobes were intimately attached by fibrous adhesions to the inner surface of the tibia, which was very much roughened by the formation in places of new osseous tissue.

The growth on the right thigh measured 17 cm. in extreme length and 14 cm. in extreme breadth. It occupied the same position over the inner surface of the tibia, and bore the same relationship to the adjacent structures as did that on the left. It was found on dissection also to be composed of three lobes, and, while these attained

larger dimensions, they were somewhat similar in shape and in structure, with the exception of the largest.

This lobe was roundish in shape and soft in consistency. Its surface was smooth. Its contents were yellowish in colour, cream-like in consistency, and had the appearance of a solution of precipitated albumen. After standing for a short time this fluid separated into two layers. The upper, which was equal to a third of the whole, was of a yellowish colour, and murky in appearance, whilst the lower was creamy in colour and solid in appearance. Numerous delicate tapering processes were attached to the inner surface of the wall of this lobe. These rarely attained 1 cm. in length, and were evidently composed of the coagulable constituents of the fluid contents. They were of a yellow colour, and resembled sebaceous matter in consistency. The examination of several cover-glass preparations, stained with gentian-violet and methylene-blue, for organisms was negative.

The tissue composing the walls of the largest lobe of the growth situated on the inner surface of the right tibia was divisible on microscopical examination—as was also possible to the unaided eye—into two layers. The tissues of these were either distinct from or blended with each other. The outer was composed of fibrous tissue. This was densely laminated in the more superficially placed parts, but it gradually assumed a looser texture. Fibroblasts were present, and were especially numerous in the looser tissues. A few blood-vessels ramified through the outer peripheral or laminated zone. The tissues of the inner layer were also divisible into two strata, but they presented no line of separation. That adjacent to the outer layer was stained brown with hæmatoxylin and eosin, and appeared granular and structureless. Throughout its substance were innumerable spaces which varied in size and communicated with one another. Fibroblasts were present in great numbers. The inner layer formed the inner surface of the lobe. It was granular, and had the appearance of being composed of small particles of coagulated albumen closely packed and joined together by connecting bands of the same material. Fibroblasts were only present, and that sparsely, in the deeper parts of this stratum.

THE WEIGHT OF THE KIDNEYS IN THE HORSE.

By J. T. DUNCAN, M.D., V.S., Professor of Anatomy,
Ontario Veterinary College.

THE records of the College in regard to the weight of the kidneys were published last year (see the *Veterinarian*, 1902, Vol. LXXV., p. 384), together with the method adopted in obtaining these records. Briefly, the method is this. Printed forms are supplied to those students assigned to a subject to be dissected. The secretary of the class, as dissection proceeds, sees that the various organs are weighed, and enters the weights on the printed form. These weights are verified by the Demonstrator of Anatomy, and the completed forms are returned to the College. These are then analysed after the close of the session, and any interesting points noted.

Predisposing.

Note first the very remarkable influence of age. It is comparatively rare that cancer occurs in young people; the majority are found between the ages of forty and sixty, most perhaps between forty and fifty. In the case of the breast, one of the commonest sites of cancer, the disease is very rare before the age of thirty, and seldom starts after seventy. So that here is a pronounced example of a predisposing cause, viz., age. Precisely why it is that tissues at this time of life are prone to cancer we do not in the least know.

Along with age there is the factor of sex, the age applying to both the sexes. The sex influence seems to be exerted mainly in determining the special position of the cancer. Thus rectal cancer is more common in men than in women, mammary cancer being most frequent in those women who have borne children and suckled them, *i.e.*, in those cases where the organ has reached full physiological development. In the uterus again the disease is almost exclusively confined to those women who have been pregnant.

Then there is the somewhat vague factor termed heredity. This is one of those phenomena whose mode of action is absolutely unknown; indeed, its existence is even denied by some, and in connection with it all that can be said is that in a certain proportion of cases there seems to be a family tendency to this or that condition, cancer being one of those conditions. It is, of course, notorious that children resemble their parents and grandparents even to trifling oddities of manner and structure, and therefore, it is argued, it is not suprising that morbid conditions and tendencies should be similarly transmitted.

Then we have such predisposing factors as are included in occupation, habits of life, etc., a somewhat vague class, but one which shows marked results in some diseases. But a very careful distinction has to be observed here between long-acting exciting causes, and merely predisposing ones. Most of the factors usually described under this head are cases in which a direct and specific influence has been acting on a person over a long period, eventually producing a diseased condition. Thus in the cancers of the scrotum which were formerly found in chimney-sweepers, it was loosely said that the patient's occupation was a predisposing cause, whereas it would be more accurate to regard the irritation of the soot as an exciting cause acting on a special tissue.

The effects of previous disease are undoubtedly to be included in the list of predisposing causes. We have noted the occurrence of cancer especially in the breast and uterus of women who have borne children. Now the common locality of uterine cancer is in the cervix uteri, which is a common seat of laceration in childbirth; and in the mammary cancers some 76 per cent. are in those who have suckled children, and a still greater percentage occurs in those who have suckled large families. Most surgeons, too, have seen cancers which have developed beneath the scar resulting from an abscess of the breast. As regards the common idea that cancer is apt to follow a blow in this region, there is very little evidence that the growth develops out of indurations and other conditions following injury. But the fact that one disease often renders the patient more susceptible to another must not be lost sight of, nor on the other hand is its

possible influence to be exaggerated (compare scarlet fever and rheumatism).

Diet has also been held to be a factor in cancer causation, some maintaining that flesh-eating is a cause of the condition, others that too frequent meals favour its occurrence, and so forth. There is but little evidence to support these statements, and at the most their influence must be of a very secondary character.

Thus, looking at the problem from the point of view of predisposition, one may say that there are certain factors such as heredity, sex, age, previous disease, etc., which undoubtedly have some influence in determining the development—especially the site of development—of cancer. It is on account of the unsearchable nature of such factors as these that modern research is confining itself to the next aspect of cancer etiology, namely,

The Exciting Causes.

The exciting causes of any morbid condition, and therefore of cancer, fall under one of three headings—they are either mechanical, chemical, or vital.

The mechanical causes include all forms of violence, internal and external, including such things as blows, falls, pressure, obstruction of tubes, both by thickening and contraction of their walls and also impaction of substances within them. Such are gall-stones, etc. They are of no importance in the present connection, because, as we saw in the case of blows on the breast, there is no evidence of causal relationship; so in the case of obstruction of tubes in the body, such as the rectum and œsophagus, they are the results, not the cause of cancer.

The chemical causes of disease include all poisonous substances whether organic or inorganic, whatever their mode of action. They therefore comprehend bacterial toxins, minerals, acids, alkalies, alkaloids, specific gland secretions, as snake venom and stings, and the various products of the liver and other glands. This class of causes may also be dismissed as having no evident bearing on cancer production.

There remain, then, among the exciting causes the ones we term vital, the class to which all the contagious or infectious diseases owe their origin, and which are specific for each disease, which are material, which pass in one or other way from those who are diseased to those who are healthy, implanting themselves in the tissues of the latter, and setting up the same morbid condition in them as was existent in the person from whom they spread. These vital causes, or living entities, grow and multiply, always reproducing the same sort of individual as those from which they sprang, though, like other creatures, they may pass through several distinct stages, requiring several hosts before they reach maturity. In this class are all the animal and vegetable parasites, and the whole group of minute organisms collectively known as bacteria. It is amongst them that so much earnest research is at the present moment being carried on with a view to demonstrating, if possible, a causal relationship between cancer and some specific organism.

Now you will doubtless be asking yourselves why I have troubled you with all this, seeing that we can find in such a review of general

etiology of disease so little to throw light on the problem. Well, it is precisely for that reason that I have done so. That is to say, a careful consideration of all these factors leads to the conclusion that there are some predisposing causes which play a part, while in the exciting causes the only one which is left open from which any result may be expected is the possibility of the disease being caused by a micro-organism.

But there is one view of the origin of disease which I have not yet touched upon, and to which I wished to lead you up by this process of exclusion. It is a view which has been advanced with special relation to the causation of tumours, concerning the origin of which we know less than that of any kind of morbid process. A good many years ago Cohnheim advanced the theory that some tumours were the result of what he termed "embryonic residues," *i.e.* certain cells in the developing embryo did not go on to form their normal future tissues, but became enclosed in abnormal situations amongst other cells, remained dormant for years perhaps, and retained during this period the capacity for developing the special tissue which they were destined to produce. In that way he accounted for the presence of hair, teeth, etc., in some morbid growths. This theory has not been applied to cancers but to other tumours, but another theory based upon a somewhat similar idea, but upon much more definite observation, has recently been brought forward in special relation to cancer. I refer to the theory of Dr Beard, of the Zoology Department, in Edinburgh University. Without going into great detail, which is no part of my present purpose, it may be stated that he accounts for the origin of cancer by supposing it to be due to the deposition of some of the products of the segmentation of the fertilised ovum in various abnormal situations in the body. These germ cells are supposed to lie dormant in these situations, until, at some later period of life, which we have seen is generally between 40 and 60 years of age, they take on growth and develop into epithelial tissue in the case of cancer. Dr Beard has seen these germ cells wandering into the situations in the body which in man are particularly associated with cancer, his observations being made upon the embryos of the skate and other lower vertebrates. The presence of these aberrant germ cells is a fact in these animals, the theoretical part of this view is that the same thing happens in the case of the human embryo, and that in man the result is the development of cancer. Now it is, of course, obvious that it will be a very difficult if not impossible task to prove this theory even if it should be the true explanation, for the simple reason that the human fertilised ovum, at the early stage in its segmentation, when these germ cells take up their abnormal situations, if they do so in man, does not come under observation. Furthermore, their existence in healthy individuals cannot be ascertained, nor their presence demonstrated. Dr Beard surmises that, as they occur in other animals, they probably do so in man, and that if so it is very likely that they take on subsequent growth in the form of cancers. This theory he communicated to the *Lancet* in a paper last year, 1902, but his paper seems to have attracted singularly little attention, and practically no criticism. I can only account for this by the fact that most medical men who are following the course of the investigations into the etiology of cancer, are doing

so from one point of view only, namely the bacteriological researches, and further, that those who did read the paper did not quite understand it. As it is written, it is by no means easy to follow for the non-zoological reader, or one who is unfamiliar with the writer's previous papers on embryological questions. But Dr Beard's theory will have to be faced by those who are enquiring into these matters, and personally it seems to me the only view which explains some of the facts.

The parasitic theory or the bacteriological theory presumes that cancer is due to the presence of some parasite or microbe, and many observations have been made in the last two or three years with a view of establishing a causal relationship between the so-called "cancer parasites" and cancer. Many observers have demonstrated the presence of round bodies in cancerous tumours, lying within and without the cells. Some of these organisms have been isolated and cultivated and experimented with, but the results have been most contradictory in the hands of different observers. Inoculation into guinea-pigs has caused a general infection with death in a month, the parasite being found on *post-mortem* in various organs of the body, with the formation of tumours consisting partly of masses of the parasite and partly of proliferated cells of the part.

The facts which seem to be in favour of a possible parasitic origin of cancer, are that the disease is most common in those glands which communicate directly with the external air or with intestinal gases, namely, the mamma, rectum, and stomach. Cancer is unusual in the thyroid and prostate. Further, the disease is common, as we saw, in the cervix uteri, less so in the uterine wall, and extremely rare in the mucous membrane of the fallopian tube. These all point to ease of infection. But a little closer consideration reveals a very great difficulty in the way of accepting the view of a bacterial origin for cancer. We define a cancer as a malignant tumour having its origin in epithelium, whether that epithelium be glandular or squamous. One must therefore suppose, on the bacterial view, that the specific organism selects those tissues only on which to act, to the exclusion of all others. But even accepting that, a very great difficulty remains. That difficulty is the extraordinary fact that the secondary metastatic growths in situations other than that where the primary cancer growth appeared are identical in structure with the primary growth, utterly independent of the tissue in which they are deposited. Cancer is structurally the same in all situations in which it occurs. What could be more remarkable than to find a secondary cancer, say in the humerus, which reproduces all the structural characteristic of a gland in the rectum? How extraordinary to find a cancerous nodule in the lung which is a secondary growth from a primary cancer in the liver, reproducing in the lung tissue the peculiar form of cancer that arises in connection with the epithelium of the bile ducts! Or, again one finds the structure of a mammary cancer developing as a secondary growth in the choroid coat of the eye, and recognises the follicles of the thyroid gland in the midst of the spinous process of a vertebra.¹ These are familiar clinical facts which militate seriously against the possibility of the growth being due to the irritating action of a specific bacillus or other parasite; but which become at least intelligible on the supposition that

¹ "Encyclopædia Medica," Vol. XII. Tumours. Bland Sutton.

the secondary growth is due to the carrying from one place to another of a germ cell which has retained its power of proliferation into epithelium; or has been there previously, and started to grow subsequently to the first growth. It is quite contrary to what happens in the case of other growths which we know to be associated with organisms. The tubercle bacillus, for example, when it infects another position from the primary one, gives rise to a growth whose intimate structure depends on the particular spot infected. So does glanders. In these infective granulomata the resulting growth is that form of granulation tissue peculiar to the organ affected, whether the growth be primary or secondary. In cancer, on the other hand, the secondary growth is literally "a chip of the old block," no matter where the secondary growth occurs. If they were due to organisms, one would naturally expect them to approximate to the type of the tissue of the new organ. It is of course conceivable that the embolus which carries the cell from the original situation carries also the micro-parasite, and that the cell multiplies in virtue of its presence, but this is pure hypothesis.

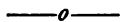
These then are the two views which at present hold the field, the parasitic and the germ-cell theory. Rather, one might say that the parasitic only is paid much attention to, for, as far as I am aware, Dr Beard is the only man in this country who is working at the subject from the embryological point of view. But his theory will have to be carefully examined sooner or later. There are several reasons why it is at present neglected. One is, that comparatively few are capable of carrying out the work necessary in that direction, I mean compared with those who are skilled bacteriologists, and whose work lies in that direction. Secondly, past bacteriological work has led to such great results from point of view of treatment, that it is natural to expect much more from it; hence the feverish search for bacterial origins of disease to the neglect of other factors. Further, and this is a factor which will operate more as the germ cell theory comes to be more recognised, if it be true, the outlook seems black indeed for the ultimate success of the prevention of cancer. For if, indeed, the origin of cancer is ultimately proved to be due to an abnormal arrangement of a few cells in early segmentation, these cells retaining their power of development in the adult body, one may well ask what hope there can be of preventing such a contingency.

Lastly, I want to direct your attention to the relative position of these two modern views to one another, a point which, as far as I am aware, has been overlooked by the exponents of both views. I speak under all reserve, for it is impossible to be absolutely up to date at any given moment on this matter, so much literature is being added every day. But as far as I know the bacteriologists have not expressed any opinion upon the way the germ-cell theory affects the bacterial, nor do I know that Dr Beard has given any indication of his opinion, if he has one, on how parasites might affect germ cells. If they exist I have not seen those criticisms. But it seems to me that herein lies the crux of the whole question. The bacteriologist says that he expects to find the cause in a parasitic organism, the biologist maintains that he has found the cause in undeveloped germ cells; is it not possible that both of them may turn out to be correct? In other

words, is it not conceivable that what one is looking for is the exciting cause, and that what the other has found is the predisposing factor? I confess that even if it were demonstrated that a specific parasite were proved for cancer, it seems to me that at the very most it could only be regarded as something which was capable of exciting certain tissues to take on growth. I cannot conceive how a parasite could be held responsible for the production of cancerous epithelial cells unless there were present in the infected animal some pre-existing cells which on receiving this specific stimulus took on the peculiar growth we know as cancer. Those pre-existing cells might be Dr. Beard's germ cells. The same argument seems to me to apply to any other exciting cause, parasitic or otherwise. If a blow or an irritant can cause cancer, it must act as an exciting cause acting upon the pre-existing potential cancer cells. Further, if this view be correct, one must suppose that the germ cells are present in a large proportion of human beings and animals, and the only reason why cancer is not more common would be that the exciting cause is relatively rare, or acts only under restricted conditions.

To sum up the position. There appears to be a certain predisposition to cancer in some families; it is certainly most common at certain ages; and it does not follow the typical mode of development, such as is found in diseases of proved organismal origin such as tubercle. On the other hand, we have certain evidence of the existence of bodies in and among cancer cells which may be parasites, and we have the fact that in lower animals some of the germ cells resulting from segmentation wander into abnormal situations in those lower animals. The two views resulting are respectively the parasitic view and the germ-cell view. While neither is as yet proved to be the sole cause of cancer, both may have their place in its etiology. If such be the case, the germ cells are probably to be regarded as the predisposing or potential cause, the fact of certain families shewing many cases being probably due to this abnormal development being more common in them: while the parasitic cause, if it exists may be regarded as the specific exciting cause, which, acting upon an already existing cell or cells, stimulates it or them to take on the growth which they should have exhibited under normal conditions in the embryo, which in the case of cancer is epithelium.

EDITORIAL ARTICLE.



THE CAUSE AND PREVENTION OF WHITE SCOUR IN CALVES.

THE interest awakened in the subject of white scour by the researches of the late Professor Nocard in Ireland during the spring of last year, has been revived by the able paper which Prof. Mettam contributed to the proceedings of the National Veterinary Association at Windermere, and which appears at an earlier part of this number. It is true that Prof. Mettam's paper is not restricted to white scour, but, in point of clinical interest and economic importance, the other morbid conditions of young or newly born animals dealt with in it are quite over-shadowed by that very common disease of young calves which has diarrhœa for its most constant and prominent symptom; and at the Windermere meeting the discussion to which the paper was subjected was mainly directed to the cause and prevention of white scour.

It will be observed that Prof. Mettam considers the cause of white scour to have been finally and conclusively elucidated by the researches which were almost simultaneously carried out by Nocard in Ireland and Lesage and Delmer in France, and he accepts the view put forward by these authors that the cause of the disease is a small ovoid bacterium of the fowl cholera type. Furthermore, he endorses the opinion that the channel by which this organism generally enters the body of the calf is the umbilical wound. The question as to what particular bacterium is the actual cause of white scour is perhaps not one of great practical importance, and, admittedly, it is, from the point of view of prophylaxis, less important than a knowledge of the way in which the agent of infection generally finds admission to the animal system. At the same time, it is undesirable that any views put forward regarding the nature of the causal organism should be accepted as final unless the evidence on which they are based is unassailable, or at least exceedingly strong. We therefore need offer no apology for calling attention to some weak points in the evidence which is held to prove that a bacterium of the fowl cholera type, or, adopting the name suggested by Lignières, a "pasteurella," is the cause of white scour.

In the first place, it is surely a remarkable fact that, although white scour is held to be a septicæmia, the particular organism which is alleged to be the cause of it appears to be rarely or never found in large numbers in the blood of affected animals; and that, even in fatal cases, its recovery in pure artificial culture from the blood or internal

organs is a matter of considerable difficulty. If it were admitted that the disease is not a septicæmia, but an enteritis analogous to human cholera, this difficulty would not occasion any surprise, or constitute a weak link in the chain of evidence ; but it is a circumstance deserving of close scrutiny when it is held that the organism in question is not essentially or mainly a bowel parasite, but one which propagates largely in the blood-stream, and is also responsible for articular and other lesions of a localised character. The extent of the difficulty may be pressed home by saying that there is certainly no other disease of a septicæmic character in which the causal organism is so constantly associated with extraneous or secondary bacteria as to make it a hard task to recover the former in a state of purity from the blood or diseased tissues. We do not forget that an attempt has been made to explain away the objection here raised by supposing that the organism of white scour generates products which have a paralysing effect on the natural defences of the body against bacterial invasions, and that, therefore, the system of a calf which is the subject of white scour soon becomes penetrated by a variety of secondary organisms which may soon come even to outnumber the original invaders. But this suggestion is just as unproved as the alleged fact which it is put forward to explain. That such secondary invasions are not uncommon in the feeble and dying is well known and generally admitted, but it may unhesitatingly be affirmed that in no known disease are such invasions so constant and extensive as to seriously interfere with the detection of the primary offender. Besides, it is worthy of notice in this connection that when animals were experimentally infected with the bacterium alleged to be the cause of white scour little or no difficulty was experienced in recovering the organism in pure culture after death. It is a little difficult to understand why in natural cases of the disease invasion by the bacterium of white scour should nearly always be followed by secondary invasions so extensive as to mask the first, whereas in animals experimentally infected with the alleged white scour organism secondary invasions are absent or inconsiderable.

To sum up the objections which may be raised with regard to this point, it may be said that we still lack evidence that this *pasteurella* is so constantly associated with cases of white scour as to compel us to believe that it must be the cause of the disease.

We have next to examine another link in the chain of evidence put forward by the discoverers of this particular organism. It is alleged that white scour can be experimentally set up by inoculating calves with the organism in question. The records of the experiments of this kind prove beyond any doubt that the bacterium employed was highly pathogenic to the calves inoculated, the animals succumbing to a septicæmic infection, with, in many cases, diarrhœa as a symptom. But it may reasonably be objected that this does not by any means prove that the bacterium producing these deadly effects is the verit-

able cause of white scour. There is a possibility of error in concluding that, when a calf infected by intravenous injection develops diarrhœa, the disease experimentally set up is white scour.

The same objection applies to the cases in which the animals were infected by the application of pure cultures to the umbilicus. It would be unfair to say that such experiments have no value. On the contrary, they would have had a high value if the evidence as to the constant presence of this organism in predominant numbers in calves suffering from white scour had been satisfactory. But, as long as that evidence is open to impeachment, the fact that inoculations with pure cultures prove pathogenic and induce diarrhœa cannot be accepted as conclusive proof that such cultures contain the organism of white scour.

The question as to what is the common method of infection in white scour is one of great practical importance. Professor Mettam subscribes to the view that the usual, if not exclusive, point of infection is the uncatrised umbilicus, and naturally one expects to have set forth the grounds on which this opinion is based. In this respect Professor Mettam's paper is disappointing. It is possible that the author relied mainly on the prophylactic success which is said to have been obtained by treatment of the navel to prove that the other possible channels of infection may be neglected. That line of argument is undoubtedly a very cogent one, and we shall return to it immediately. It must be remembered, however, that the umbilicus was proclaimed to be the point of infection before the prophylactic effect of asepsis of the navel had been proved in practice. The belief that the umbilicus was the common port of entrance of the organism was founded on the fact that calves could be infected and killed, with symptoms and lesions more or less closely resembling those of white scour, by injecting pure cultures of the organism alleged to be the cause of white scour into the veins, or applying them to the navel, while attempts to infect by administering similar cultures by the mouth failed. Obviously, however, this line of argument falls to the ground if, as we have already shown, there is a flaw in the evidence relied upon to prove that the cultures employed were those of the bacterium of white scour.

If one desires to ascertain whether calves can be infected with white scour by way of the alimentary canal, the proper method of procedure is not to employ cultures of an organism which possibly may have nothing to do with the genesis of the disease, but, at least in the first instance, to try whether the diarrhœic discharges or the diseased tissues of calves affected with white scour are capable of communicating the disease when they are administered by the mouth.

It will be noted with some surprise that, although Professor Mettam regards the umbilicus as the common point of infection,

he quotes without dissent experiments by Jensen, who "obtained infection in seven calves by feeding them on milk mixed with intestinal discharge of animals suffering from the disease." No one is entitled to deny that infection by the mouth is possible, and even the ordinary way in which the disease is contracted, unless he has himself experimented as Jensen did with negative results, or is prepared to show that there was some fallacy in Jensen's experiments.

As long as Jensen's experiments and conclusions are uncontroverted, it must be considered highly probable that most cases of white scour are due to infection by way of the alimentary canal.

Lastly, the practical value of navel treatment in preventing white scour, and the support which this lends to the view that infection usually takes place by way of the umbilicus, must be examined. Professor Mettam says, "in my opinion prevention is to be attained by careful attention to the umbilicus." But, somewhat inconsistently, he adds, "but no matter how careful we may be in the umbilical treatment, this must be supported by cleanliness in and about the houses inhabited by the young and their dams. Treatment of the umbilicus may save animals; there is no doubt that thorough hygienic surroundings will cut down the mortality; both should theoretically remove altogether the cause of death. This has been our experience in Ireland."

It appears to be permissible to suggest that, if this second quotation is to stand, the first should read, "prevention is *not* to be obtained by careful attention to the umbilicus." It may also be suggested that, if the umbilicus is the exclusive port of entrance of the germs of the disease, careful navel treatment by itself should, theoretically, altogether remove the cause of death. On the other hand, if in a large proportion of cases infection takes place by the mouth, it would be very rash to say that navel treatment, even when it is carried out in "thorough hygienic surroundings," will altogether remove the cause of death—that is, assuming that the intestinal discharges of diseased calves contain the agent of infection. However, it will be observed that, whatever one might have been entitled to expect theoretically, Professor Mettam's experience has been that the cause of the disease has been altogether removed in Ireland by combining navel treatment with strict attention to the hygiene of the byres and calf-houses. One could heartily wish that this experience had been universal, but, unfortunately, it does not appear to have been general even in Ireland.

In the course of the present year¹ a report has appeared setting forth the results obtained on a number of selected farms on which the calves were treated in accordance with the recommendations

¹ "Journal of the Department of Agriculture and Technical Instruction for Ireland," March 1903.

made by the late Professor Nocard, the navel treatment being also supplemented by thorough disinfection of the byres and calf-houses. On a number of the farms the treatment was carried out under the supervision of a person "sufficiently skilled to direct operations, and to ensure to the treatment a fair trial." At the remaining farms the carrying out of the treatment was entrusted to the owners.

The following table is taken from the report :—

<i>Name of Centre.</i>	<i>No. of Calves treated by Owners.</i>	<i>No. died.</i>	<i>No. treated under Supervision.</i>	<i>No. died.</i>	<i>No. treated under partial Supervision.</i>	<i>No. died.</i>
Bansha	6	4	22	7	19	8
Feenagh	6	4	16	6	6	—
Murroe	7	1	17	2	—	—
Newcastle West	1	1	20	8	2	2
Liscarrol	18	8	22	15	—	—
Bunratty	—	—	49	2	2	1
TOTAL	38	18	146	40	29	11

The comment which the writer of the report makes upon the results indicated by the figures in the above table is that they are "far from satisfactory." Moreover, the leaflet which the Irish Department of Agriculture is now issuing for the guidance of farmers contains the statement that, "Navel treatment without repeated and absolute disinfection will NOT be successful," and concludes with the warning, "The main point in prevention is disinfection of the premises. Half measures will not be effective."

In face of these admissions, it is not possible to agree with Professor Mettam's optimistic estimate of the beneficial effects of navel treatment, even when it is supplemented by thorough cleansing and disinfection of the premises. It is also impossible to extract from the results obtained in Ireland any support for the view that the disease is always or generally umbilical in its starting-point. On the contrary, they strengthen the opinion that umbilical infection is not responsible for more than a minority of the cases of so-called white scour, and that diarrhœic discharges are the vehicle, and the mouth the usual port of entrance, of the agent which is the cause of the disease.

We do not pretend to know what the author of the Irish leaflet from which we have quoted means by absolute disinfection, nor can we conceive how anything deserving of the name can be obtained in an inhabited calf-house. We agree, however, that isolation of the already affected calves, combined with repeated and thorough cleansing and disinfection, must be made the keystone of preventive measures. Unfortunately, it cannot be said that, even theoretically, these measures ought always to succeed in arresting the spread of

the disease. In this connection it must be remembered that a diseased calf is one which harbours the germ of white scour, and that, just as with many other bacterial diseases, this abnormality may not be manifested by any pronounced symptom of disturbed health. Hence, in any outbreak of white scour it may be practically impossible to distinguish with certainty between the infected and the non-infected animals, although that is precisely what is of cardinal importance when one wishes to arrest the spread of the disease. In short, the difficulty in preventing white scour is just the same as that with which we have long been familiar in the case of other contagious diseases, such as glanders, tuberculosis, and pleuro-pneumonia.

CLINICAL ARTICLES.

A CASE OF CYSTIC CANCER OF THE LIVER IN A SHEEP.

By J. F. HODGSON, M.D., Ch.B.Vict., Deputy Medical Officer of Health, Halifax.

Cystic cancer of the liver is an exceedingly rare form of disease in man,¹ while cancers of any variety are uncommon in cattle and sheep.²

In the public slaughter-house at Halifax over 17,000 sheep are killed annually, yet during the last four years, for certain, only in one case has cancer of the liver been found. It must be borne in mind, however, that the chances are against a sheep with such a rapidly fatal disease ever reaching the slaughter-house, but still, I think, the figures show the great rarity of the disease.

In the case under notice the liver weighed 17 lbs., and was enormously enlarged. Only a very small portion of liver tissue was left, and this had on the surface the appearance of a coarse cirrhosis. The rest of the surface presented a very irregular bossy appearance. On section the small portion of liver tissue was seen to be cirrhotic and slightly fatty, while the rest of the organ was made up of nodular growths and cysts. In fact, the general appearance of the liver would almost lead one to think that it was composed of little else but cysts in a fibrous network. The newer growths were of a greyish yellow colour, of fairly firm consistence, and generally globular in shape. When these nodules got beyond the size of a cherry softening occurred, leading to the formation of cysts, which contained blood and debris. The cancerous walls of the cysts were well marked, and the spaces between

¹ Coats "Manual of Pathology," pp. 906, 907. Hale White "Tumours of the Liver in Allbutt's System of Medicine," vol. iv., p. 211.

² Walley's "Meat Inspection," p. 53.

them and the blood clots contained a thickish, slightly blood-stained fluid, which escaped on section.

The gall bladder and the bile ducts were free, but the glands in the portal fissure were affected.

The carcase was very little emaciated, and showed no signs of jaundice.

A microscopical section through a small nodule showed typical "cell-nests," epithelial in character, enclosed in a well-marked vascular network of fibrous tissue.

The entrails had been destroyed, so that it was impossible to make a careful examination for cancerous deposits in other organs. The liver is the organ most liable to secondary cancer, and the probabilities are that in this case the growths were of that nature.

I have to thank Mr J. K. Crawshaw, our Meat Inspector, for bringing this case to my notice.

MAMMARY TUMOUR IN A SOW.

By A. G. HOPKINS, B.Agr., D.V.M., Vancouver, B.C.

DURING the weaning period the mammary gland of a pure-bred Yorkshire sow was noticed to be increasing instead of decreasing in size at the posterior part. The use of a home-made irritant dressing tended to reduce it temporarily to the size and hardness of a baseball. As the sow approached another parturition, the gland again enlarged to a very great size, and again after parturition was over subsided to some extent, although it did not become as small as at the weaning period first mentioned.

The sow was approaching a parturition when the writer's attention was first called to her; therefore Nature was allowed to take its course, and eleven pigs were delivered. Their subsequent deaths were due to other causes, I believe, than the presence of the tumour.

The tumour impeded the sow's movements to a considerable extent, and its under surface became abraded from friction with the ground (as shown in the photograph). Six weeks after the parturition, removal was attempted with the knife and *écraseur*. The tumour on section showed a considerable amount of very firm tissue, enclosing many large pockets of an ill-smelling pus; the growth was well supplied with blood by one main vessel. The weight of the tumour after removal was estimated to be about 30 lbs.

After the edges of the skin had been brought together by sutures (an overplus of skin had to be removed) the sow was let up and was able to run around the barnyard, although stepping high with the hind legs, which had not become accommodated to the loss of the tumour.

The operators looked carefully over the wound site for fugitive portions of the growth, but were unable to find any. They were endeavouring to be as conservative as possible in the removal of the gland and teats, and deemed the growth completely excised, but subsequent events showed they were mistaken.

The coldness of the atmosphere (winter) and the surroundings

possible influence to be exaggerated (compare scarlet fever and rheumatism).

Diet has also been held to be a factor in cancer causation, some maintaining that flesh-eating is a cause of the condition, others that too frequent meals favour its occurrence, and so forth. There is but little evidence to support these statements, and at the most their influence must be of a very secondary character.

Thus, looking at the problem from the point of view of predisposition, one may say that there are certain factors such as heredity, sex, age, previous disease, etc., which undoubtedly have some influence in determining the development—especially the site of development—of cancer. It is on account of the unsearchable nature of such factors as these that modern research is confining itself to the next aspect of cancer etiology, namely,

The Exciting Causes.

The exciting causes of any morbid condition, and therefore of cancer, fall under one of three headings—they are either mechanical, chemical, or vital.

The mechanical causes include all forms of violence, internal and external, including such things as blows, falls, pressure, obstruction of tubes, both by thickening and contraction of their walls and also impaction of substances within them. Such are gall-stones, etc. They are of no importance in the present connection, because, as we saw in the case of blows on the breast, there is no evidence of causal relationship; so in the case of obstruction of tubes in the body, such as the rectum and œsophagus, they are the results, not the cause of cancer.

The chemical causes of disease include all poisonous substances whether organic or inorganic, whatever their mode of action. They therefore comprehend bacterial toxins, minerals, acids, alkalies, alkaloids, specific gland secretions, as snake venom and stings, and the various products of the liver and other glands. This class of causes may also be dismissed as having no evident bearing on cancer production.

There remain, then, among the exciting causes the ones we term vital, the class to which all the contagious or infectious diseases owe their origin, and which are specific for each disease, which are material, which pass in one or other way from those who are diseased to those who are healthy, implanting themselves in the tissues of the latter, and setting up the same morbid condition in them as was existent in the person from whom they spread. These vital causes, or living entities, grow and multiply, always reproducing the same sort of individual as those from which they sprang, though, like other creatures, they may pass through several distinct stages, requiring several hosts before they reach maturity. In this class are all the animal and vegetable parasites, and the whole group of minute organisms collectively known as bacteria. It is amongst them that so much earnest research is at the present moment being carried on with a view to demonstrating, if possible, a causal relationship between cancer and some specific organism.

Now you will doubtless be asking yourselves why I have troubled you with all this, seeing that we can find in such a review of general

On arrival I found her being led about in the yard, but the pain had left her, though she was still going lame on the near hind, and the muscles covering the tibia (near) appeared to be slightly swollen. I ordered her to be put into the stable, and the leg to be fomented, and did not see her again until the 4th April, but the horsekeeper informed me that she regained free use of her leg in four days, and was allowed to stay in a box until the 4th April, when she was sent out to work. This time she did not go a quarter of a mile before the same symptoms again came on, but more severely. She was walked back to the stable and put in a loose box. She was in great pain and could not be kept on her feet, kept rolling on her back and kicking. Again I did not see her until about three hours after the onset of symptoms. On arrival I found her free from pain, but moving rather stiffly on her hind-legs, and I concluded that she must have had a slight attack of azoturia, as she had been taken straight from resting to work. I gave her a physic ball, and ordered her to have a little walking exercise after the physic had operated, before putting her to work.

About the 14th April I met the horsekeeper, who informed me that on taking her out for walking exercise she very soon began to knuckle at the hind fetlocks, and that he was glad to get her back into the loose box. I told him to persevere with the exercise, and that I should like to see her when showing these symptoms.

On the 20th April I was sent for in great haste, as she was worse than ever before. She had been led out, and had made a circle of a quarter of a mile. This time I arrived about half an hour after the onset of the attack, and found her down in the box, unable to rise, in great pain, rolling on her back, sweating all over the body except the hind-legs, respirations 20, pulse 60, temperature normal, urine normal in colour. After about half an hour I managed to get her on her feet with a little help from a couple of men at her tail and a good support from the head collar, but she could not stand on her hind-legs, and went down in about two minutes. I gave her an anodyne draught, and fixed her head so that she could not knock it up against the wall.

I informed the owner that she was useless, as I was confident of thrombosis of an iliac artery (near), and would be glad if he would give me an opportunity of making a *post-mortem*, which he did four days after. During the four days he kept her, she did not stand up more than two hours altogether.

The *post-mortem* examination showed thrombosis of the left internal and external iliac arteries, and of the right internal iliac artery. The termination of the aorta, between the roots of the external iliacs, was also occupied by thrombus material, continuous with that in the above-named branches. These three branches appeared to be completely obstructed.

In conclusion it may be added that while resting in the stable from 16th March to 25th April the mare gained rapidly in flesh, and when walked in the yard she would kick and jump on her own account.

Reports.

PROFESSOR KOCH ON RHODESIAN REDWATER OR AFRICAN COAST FEVER.

INTERIM REPORT.¹

I have the honour to submit herewith my first report embodying my observations and investigations with regard to the cattle disease existing in Rhodesia, in response to your telegraphic request of the 17th March.

Having received a letter from the British South Africa Company on 24th December 1902, authorising me to proceed to Rhodesia, via the East Coast. I left Berlin with my two assistants, Dr Neufeld and Dr Kleine, Army Medical Department, on 12th January 1903, and landed at Beira on 18th February. On the way I availed myself of the opportunity to make investigations and inquiries at the various ports at which the steamer called along the East African Coast, at Mombassa, Tangar, Zanzibar, Dar-es-Salaam, Kilwa, Ibo, Mozambique, and although the stoppages were brief I was able to collect valuable material throwing much light on the subject of the Rhodesian cattle disease, to which I will refer later.

In Beira I was met by Mr Gray, Chief Government Veterinary Surgeon of Rhodesia, and Col. Beal, Assistant Manager of the Beira and Mashonaland Railways, who kindly arranged everything to enable me to proceed without delay to Umtali.

Mr Gray gave me every information about Beira, and about the first outbreak amongst the imported cattle from New South Wales at the end of 1900.

On the afternoon of the day on which we arrived we visited the farm of Mr Martini, outside Beira, and inspected his cattle, which were of interest inasmuch as they had grazed alongside the Australian cattle and sometimes even mixed with them.

We then proceeded to Umtali, where we arrived on 20th February. Here we saw the first case of the disease in a calf, the survivor of a large herd, but we could carry on no further investigations here, as the disease had spent itself, and only a very few head of the cattle were left.

A similar state of affairs existed in Salisbury, where we also saw some cases, but, as at Umtali, the epidemic, after having killed off nearly the whole stock of the township, had temporarily abated.

This being the case, we therefore decided to proceed to Bulawayo, where the disease had broken out only some months previously, and where there is every likelihood that for some time to come suitable material for the study of the disease will be found.

The Hillside Camp, $1\frac{1}{2}$ miles outside Bulawayo, which was erected two years ago for the accommodation of the Rhodesian Field Force, but which is now unoccupied, affords an excellent site for a laboratory and for housing experimental animals, although some alterations are necessary to prepare stables in which healthy animals can be kept without danger of infection, by no means an easy matter in such a highly infected neighbourhood. I expect, however, the work of carrying out these alterations will be so far advanced in the course of a few days, under the expert supervision of Mr Gray, that experiments with healthy animals, with a view to conferring immunity, can be commenced by placing as many infected animals as possible under observation.

¹ This report is dated March 26th 1903.

Predisposing.

Note first the very remarkable influence of age. It is comparatively rare that cancer occurs in young people; the majority are found between the ages of forty and sixty, most perhaps between forty and fifty. In the case of the breast, one of the commonest sites of cancer, the disease is very rare before the age of thirty, and seldom starts after seventy. So that here is a pronounced example of a predisposing cause, viz., age. Precisely why it is that tissues at this time of life are prone to cancer we do not in the least know.

Along with age there is the factor of sex, the age applying to both the sexes. The sex influence seems to be exerted mainly in determining the special position of the cancer. Thus rectal cancer is more common in men than in women, mammary cancer being most frequent in those women who have borne children and suckled them, *i.e.*, in those cases where the organ has reached full physiological development. In the uterus again the disease is almost exclusively confined to those women who have been pregnant.

Then there is the somewhat vague factor termed heredity. This is one of those phenomena whose mode of action is absolutely unknown; indeed, its existence is even denied by some, and in connection with it all that can be said is that in a certain proportion of cases there seems to be a family tendency to this or that condition, cancer being one of those conditions. It is, of course, notorious that children resemble their parents and grandparents even to trifling oddities of manner and structure, and therefore, it is argued, it is not surprising that morbid conditions and tendencies should be similarly transmitted.

Then we have such predisposing factors as are included in occupation, habits of life, etc., a somewhat vague class, but one which shows marked results in some diseases. But a very careful distinction has to be observed here between long-acting exciting causes, and merely predisposing ones. Most of the factors usually described under this head are cases in which a direct and specific influence has been acting on a person over a long period, eventually producing a diseased condition. Thus in the cancers of the scrotum which were formerly found in chimney-sweepers, it was loosely said that the patient's occupation was a predisposing cause, whereas it would be more accurate to regard the irritation of the soot as an exciting cause acting on a special tissue.

The effects of previous disease are undoubtedly to be included in the list of predisposing causes. We have noted the occurrence of cancer especially in the breast and uterus of women who have borne children. Now the common locality of uterine cancer is in the cervix uteri, which is a common seat of laceration in childbirth; and in the mammary cancers some 76 per cent. are in those who have suckled children, and a still greater percentage occurs in those who have suckled large families. Most surgeons, too, have seen cancers which have developed beneath the scar resulting from an abscess of the breast. As regards the common idea that cancer is apt to follow a blow in this region, there is very little evidence that the growth develops out of indurations and other conditions following injury. But the fact that one disease often renders the patient more susceptible to another must not be lost sight of, nor on the other hand is its

possible influence to be exaggerated (compare scarlet fever and rheumatism).

Diet has also been held to be a factor in cancer causation, some maintaining that flesh-eating is a cause of the condition, others that too frequent meals favour its occurrence, and so forth. There is but little evidence to support these statements, and at the most their influence must be of a very secondary character.

Thus, looking at the problem from the point of view of predisposition, one may say that there are certain factors such as heredity, sex, age, previous disease, etc., which undoubtedly have some influence in determining the development—especially the site of development—of cancer. It is on account of the unsearchable nature of such factors as these that modern research is confining itself to the next aspect of cancer etiology, namely,

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that cattle from Natal and the Transvaal, which are, as we know, immune against Texas fever as existing in Natal and the Transvaal, and even cattle from Texas itself, will catch the Rhodesian fever and die of it. Were the two diseases of exactly the same character, a complete or at least a certain degree of immunity should have been manifested, but this is not so, therefore they cannot be considered identical, nor is the view that Rhodesian fever is merely a more than usually virulent form of Texas fever a tenable one.

Yet another theory has been propounded, and that is that the Rhodesian fever is not one disease, but a complication of diseases; but in support of this there is no evidence whatever. In the blood of animals examined by us we only found the small parasites characteristic of this disease, and always in such abundance that the course of the disease and the death of the animals could be ascribed to their presence without any hesitation, and there was no necessity to entertain any theory of the existence of any mixed infection.

The question is now if this apparently new disease has arisen in Rhodesia, or if it was imported from some other place, and to answer this question I must endeavour to trace the history of this outbreak.

At the end of 1900 about 1000 head of cattle for Rhodesia from New South Wales were landed at Beira, and, because they could not be forwarded by rail at once, they were sent on the veldt for grazing there. They grazed in the neighbourhood of Mr Martini's farm and mixed with his stock, which were to all appearances in perfect health, and which remained so. Two or three weeks later the disease broke out amongst the Australian cattle, which were then sent to Umtali, and carried the disease with them.

But how could the Australian cattle become infected? In New South Wales no such disease is known, and no mortality occurred amongst these animals during their voyage from Australia. There was also no apparent disease at Beira.

The problem would be well-nigh insoluble if there was not one piece of evidence in our possession which I became cognisant of some years back—namely, that the same disease exists on the East African coast as has now broken out in Rhodesia, and that in German East Africa the disease was carried inland in a similar manner to that in which it may have been brought from Beira to Umtali.

The stock on the coast of German East Africa are apparently quite healthy and in the best condition, but as soon as other cattle are brought there from clean districts—for instance, from the inner parts of the country—and graze on veldt on which the cattle from the coast have been grazing, these imported animals will get sick and almost all die.

The indications of this disease are the same as those of Rhodesian fever, and in the blood of these infected animals, which I have many times examined, I have always found the same small blood parasites which I have again seen at Umtali, Salisbury, and Bulawayo.

On the voyage out I have had an opportunity of examining three animals which only a few weeks previously arrived at the coast of German East Africa from the interior, and which were showing the first symptoms of the disease, and whose blood contained the characteristic blood parasites present in Rhodesian fever. My assistants also found the same parasites in the blood of several sick animals at Zanzibar. Six years ago, at Tanga, I saw an outbreak of exactly the same character as the present Rhodesian one, although of smaller dimensions. In this case a herd which was brought into the infected area at Tanga, and which became infected there, carried the disease thence to Mohesa in the interior.

From inquiry which I made, I gathered that this disease has been indigenous for generations back in German East Africa and the neighbouring islands, and I have good reason for supposing that it also extends still further

southwards along the coast, and in all probability it also exists in Beira. In Beira I was informed that some time ago cattle were frequently brought there from German East Africa and Madagascar, and that the latter animals, especially when they came from the south of the island, soon became sick and died, while the cattle from the East African coast and the northern districts of Madagascar remained healthy.

To verify these statements, to see if the cattle from German East Africa and from Beira are immune against Rhodesian fever, which should be the case if my views are correct, I consider it is well worth while bringing a few cattle from German East Africa and from Beira to Bulawayo, and to be tested here as to their immunity.

In conclusion, it seems to me a very reasonable explanation of the cause of the present Rhodesian outbreak is to assume that the highly susceptible Australian cattle landed at Beira, which belongs, I believe, to the infected coast district, became infected there, and carried the disease with them to Rhodesia.

I am of opinion that there is also good reason for supposing that the infected area extends still further south to Delagoa Bay, as for some time past the Transvaal has been invaded from its south-eastern border by a slowly spreading disease infecting cattle, which appears from all accounts to be very much like the Rhodesian fever. M. Theiler, Government Veterinary Bacteriologist, Pretoria, whom I asked to send me some blood preparations from infected parts of the Transvaal, most kindly complied with my request, and we have been able to prove that in the preparations from different parts of the Transvaal, (from Nelspruit, Herdepoort and Mooi Plaatz) exactly the same blood parasites are to be found as at present in the blood of animals affected with local Rhodesian fever; therefore, there can be no doubt that the same disease exists in the Transvaal as that which prevails in Rhodesia, but when we take into consideration the position of the infected districts of the Transvaal, and the obstacles to communication which have existed and still exist, the view that the disease extended thither from Rhodesia is, I think, quite untenable. What is very likely, however, is that the disease obtained a foothold in the Transvaal in much the same way as it did in Rhodesia, that is, by being imported from the coast, in the one instance from Beira, and in the other from Delagoa Bay. In appearance in Rhodesia the disease has conformed to a natural law which it has often followed aforetime. It has been brought inland from the infected coast districts as it has previously been in many other instances, and therefore it appears to me more reasonable to call it "African coast fever," in preference to calling it "Rhodesian fever."

Taking these facts into consideration, it is advisable that those ports on the East African coast which are now used as ports of entrance for the introduction of cattle to the interior should be carefully examined in order to ascertain whether or no they are infected with "African coast fever," and those which are so infected should be closed against cattle, or the entrance of these animals should only be permitted under such conditions that the importation of infection from the coast to the interior is rendered impossible.

Another important question which arises in connection with the present epidemic is whether anything like lasting immunity against the disease exists in such animals as have recovered from an attack of the disease. It is known that immunity against Texas fever exists, but whether there is any lasting immunity to the Rhodesian fever seems doubtful, as in the report of Gray and Robertson they state, "the few animals which have recovered from a first attack in many instances developed a second attack about three months later which terminated fatally," and we also saw a case in Salisbury which ended in the death of the animal, which we were assured had got over the disease some months ago. This does not look as if we could reckon upon immunity, but still I am convinced that such a thing exists, because we have had an

opportunity of seeing and examining a considerable number of animals which have recovered from the disease, and although they have been grazing for some time on infected veldt they have not sickened again, and must therefore be considered immune against natural infection.

A most interesting feature of the examination of these animals was, that although they appeared perfectly healthy, and did not show the slightest variation in temperature, their blood contained a small number of parasites, which can only be demonstrated satisfactorily by the use of a staining reagent (Azur. 11) which, like the Romanowsky method of staining for malaria, colours the chromatin in the parasites a dark red. By this staining method every single parasite can be identified with certainty, and it is to be hoped that in this manner immune animals can be easily and certainly recognised. An immunity of this description against Rhodesian fever, which is characterised by the presence of a small number of parasites in the blood, resembles very closely that existing against Texas fever, as here, too, parasites exist in a certain number in the blood of immune animals, as the reaction following inoculation with their blood proves. The existence of such parasites in the blood of an apparently healthy animal may be taken as an indication that such animal is not inconvenienced by their presence, and therefore must be immune.

As we know that in the pyrosomal diseases, whereto the Rhodesian fever belongs, ticks play a most important part as agent for the dissemination of infection, I have occupied myself thoroughly with this side of the question. For this I have had special facilities, as in my earlier work on the pyrosomal disease at Dar-es-Saalam, which I have identified with Rhodesian fever, I reproduced it with infected ticks in the far distant Usambara Mountains, and so proved that the ticks are also the intermediary agents in this disease.

Putting on one side a few rare species of ticks, I have found the cattle ticks of Rhodesia to be of the same species which are described commonly as throughout South Africa. They are the following:—

Rhipicephalus decoloratus, *rhipicephalus evertsi*, and *hyalomma ægyptium*, and to these we must add *rhipicephalus sanguineus*, which is the most common tick in Rhodesia. In Umtali we found also *amblyomma variegatum* frequently on cattle, and now and then we found *hæmaphysalis leachi*, of which the dog is the usual host.

Of real importance amongst these ticks there are only the different species of *rhipicephalus*, and of these most particularly the *rhipicephalus decoloratus* or blue tick, because this insect is most closely allied to ticks which in America (*rhipicephalus annulatus*) and in Australia (*rhipicephalus australis*) carry the Texas fever, and also because it is known to be the tick which disseminates Texas fever in South Africa. For this reason we may suspect that *rhipicephalus decoloratus* is also the intermediary agent in Rhodesian fever. Up to now there have been no direct experiments made, and there is a possibility that the other two species of *rhipicephalus* may also act as carriers. Experiments have already been begun to decide this question.

With regard to the *rhipicephalus decoloratus*, it is known that it differs from the American and the Australian species in possessing only six rows of teeth on its sucking apparatus (labium), instead of the eight rows common to both the others. Up to now the general opinion was that the area for the *rhipicephalus decoloratus* on the East African coast extended far to the north; I therefore made a collection of ticks at the various ports of call on my journey down the coast, and found that from Mombassa to Beira, with the exception of the Island of Ibo, everywhere specimens of *rhipicephalus* were obtained, and amongst these a kind which would appear to a casual observer to resemble the *rhipicephalus decoloratus*, but which on more careful examination was found to have eight rows of teeth, and is therefore more closely related to the American and Australian varieties than the African *rhipicephalus decoloratus*. This specimen is found all along the east coast of Africa as far south as Beira,

and I suspect it may also exist in Delagoa Bay and beyond. Of the real *rhhipicephalus decloratus* I have not found a single specimen on the coast, but in Rhodesia, as far as my investigations went, only the *rhhipicephalus decloratus*, with six rows of teeth, exists. Nor have I found in Rhodesia any specimens of the eight rowed coast species. This fact goes to prove that in carrying the disease from Beira to Rhodesia the Australian cattle only brought to Umtali the parasite which produces the disease, and not the coast tick which carried it; but, unfortunately, it turned out that there was already in Rhodesia a variety of tick, probably the *rhhipicephalus decloratus*, quite as capable of acting as a carrier as is the coast *rhhipicephalus*.

Lastly, I come to the question of how the disease will further develop, and in what way we can combat its spread. If nothing could be done against the disease, it would, without any doubt whatever, permanently infect every locality in which it breaks out, and in time the whole of Rhodesia will suffer as severely as Umtali, Salisbury, and other places have already done.

There will be only 10 to 20 per cent. of the cattle left alive, but these animals will be immune, and will be progenitors of an immune breed which will thrive in Rhodesia as they now do on the coast. The same situation will be developed as now exists in the southern states of North America, in Queensland, and on the East African coast; the locally bred cattle will not become visibly sick, they may appear strong and healthy, but the progeny of ticks reared upon such animals will be pathogenic for cattle which come from clean countries. This state of affairs can hardly be considered a satisfactory one. Besides this, there is to be feared that this natural immunising process will progress very slowly and fitfully, and as a result the cattle breeding industry in Rhodesia will not develop materially for many years to come. As this is the alternative, it is absolutely necessary not to let the pest take its own course, but to fight against it by every means in our power. A radical measure would be the destruction of all ticks, but this has been attempted for years in the Texas fever countries, but unfortunately without marked success; because, while it is possible with the help of dipping tanks and sprays to destroy most of the ticks on an animal, some remain, as we have noticed on a few regularly dipped cattle here, and very few ticks will suffice for the production of a large number of young ticks which will ensure further spread of the disease.

Therefore, experts in North and South America, as well as in Australia, came to the conclusion that it would be well to protect the animals against the sickness by inoculation. At first these inoculations were not always satisfactory, as sometimes the blood used for inoculation purposes was too weak to protect, or too virulent. It was only when blood could be obtained from such immune animals as gave a sufficiently severe reaction to confer immunity, and when this standard of virulence could be maintained, that produced satisfactory results were forthcoming. Such a line of action must be adopted to combat Rhodesian fever, and our endeavour must be to find a suitable virus—a matter of no little difficulty on account of the extreme virulence of the Rhodesian fever; and when once the fact is ascertained that natural immunity to this disease actually exists, there is no doubt the line of treatment necessary to produce immunity artificially will be forthcoming sooner or later.

What remains to be done is to discover a standard virus of such strength that it will confer a maximum immunity with a minimum of loss from inoculation. Should such a virus be unobtainable by any other means, then an effort must be made to attenuate the virulence of the parasite, for example, by passages through animals other than cattle, or in some other method by which we may hope to procure the desired protection. That this is possible is shown by the fact that the experiments which I have formerly made in East Africa accidentally placed in my hands a method of immunisation to which I

subjected certain cattle which subsequently resisted a natural infection when exposed thereto.

Of course it is my intention to do my utmost to obtain a suitable stock vaccine as soon as possible.

I will experiment simultaneously in many different lines in order to save time, and for the purpose many healthy cattle will be required.

The more animals we have for this experimental work the sooner we may hope to bring our labours to a satisfactory termination.

SECOND REPORT.¹

In presenting this, my second report on African Coast fever, allow me to supplement it by stating that at this stage of our work I cannot furnish what may be looked upon as a complete record of results, but I can only indicate the direction in which our researches are being prosecuted, how they proceed, and at what stage they have arrived. Experiments in African Coast fever require much longer than rinderpest experiments. In rinderpest negative or positive experimental results are forthcoming in every case in from one to two weeks, whereas in African Coast fever four weeks are necessary, as the incubative period of the disease is still uncertain, and in the case of tick experiments the time taken up is even longer, one or two months being occupied in hatching out the ticks, and one month more elapsing before the experiment can be considered concluded—this brings the total period necessary for tick experiments up to two or three months.

Soon after sending in my first report a series of experiments on healthy animals were inaugurated. For these experiments our animals were drawn from the Plumtree District, in the South of Rhodesia, which is at present free from disease. Our healthy animals were forwarded by rail from Plumtree and detained in Bulawayo, whence they were driven by road to Hillside Camp. On their arrival there, to prevent any likelihood of accidental infection *en route*, each animal was well sprayed with a 25 per cent. solution of paraffin in water, and then all were placed in buildings enclosed by a wire fence surrounding these at a distance of four yards. To prevent any possibility of ticks being harboured within the enclosure, all vegetation and grass has been removed within the fence, and the cattle are fed upon imported forage brought from Kimberley, a disease-free area. That these precautions are a sufficient protection is shown by the fact that no case of extraneous infection has occurred amongst these animals since they arrived in Bulawayo.

On the arrival of each lot of healthy animals, their blood was submitted to a preliminary microscopical examination. This examination revealed the remarkable fact that the blood of many of them contained the ordinary pear-shaped micro-organism of common Texas fever or redwater, indicating that in the Plumtree district ordinary Texas fever is an endemic disease.

Amongst the first batch of Plumtree animals received, numbering eighteen, pear-shaped organisms were present in no fewer than eight cases, although all the animals were apparently in perfect health, indicating that their infection with the disease was not of recent date, but was rather of long standing. This fact has an important bearing upon the whole investigation, and must be taken into consideration in our further work.

Some experiments made with the blood of these animals which contained the micro-organisms of ordinary redwater, and data furnished me by Chief Veterinary Surgeon Gray, prove conclusively that ordinary redwater infection has existed along the main transport roads throughout Rhodesia, between Bulawayo and the South, between Bulawayo and Salisbury, and along other trade routes, for a long time, and therefore every ox which has worked along

¹ This report is dated 28th May 1903.

these roads for any lengthy period may be reasonably suspected of harbouring in his blood the organisms of redwater.

Now, we know that animals of this class, living in a region where Texas fever is endemic or which have come therefrom, may have relapses of this disease. These relapses are indicated microscopically by the appearance of numerous Texas fever organisms in their blood, and are most likely to occur if such animals are attacked by any other febrile disorder by which their vitality is lowered, as such a lowering of their vitality gives the Texas fever organisms a chance to multiply. An experience of this sort is not uncommon in rinderpest, as it has been found that when an animal immune to Texas fever and retaining in its blood the organisms of that disease is attacked by rinderpest, it may develop not only rinderpest but at the same time Texas fever. Much slighter causes than attacks of a serious disease of this class may induce relapses in specially predisposed animals which have at some previous time suffered from redwater, mere elevation of temperature, such as may result from overwork, sometimes being sufficient.

This being our experience, it might be expected that such predisposed animals which had previously suffered from redwater might again show symptoms of it if attacked by African Coast fever; and, starting with such a hypothesis, a very simple explanation is forthcoming for certain phenomena which have been observed during the present epidemic, such as the presence of large pear-shaped organisms in the blood in certain cases, and the occurrence of hæmoglobinuria. These pear-shaped parasites are not found in all cases of African Coast fever, nor do they appear simultaneously with the bacillary organisms, but they show themselves in the later stages of the disease, while the hæmoglobinuria which we have observed occasionally we have only seen in cases in which the pear-shaped organisms, hitherto considered characteristic of Texas fever, have been found along with the organisms of the bacillary type.

In all, we have examined the blood of ninety-one sick animals. In every instance we have found the small parasites of African Coast fever, but only in ten cases have we found these parasites in conjunction with the larger pyriform organisms, and in six of the latter cases we have observed blood-coloured urine.

These observations bear out the view that amongst the animals whose blood we have examined there were a certain number of specially predisposed animals salted to ordinary redwater, which again developed this disease as a result of the high temperature produced by an attack of African Coast fever.

Evidence in support of this theory can also be adduced from a consideration of the outbreak which carried off the imported Australian cattle. Here we had animals susceptible to, and exposed to, infection both with redwater and African coast fever. Probably some were more susceptible to ordinary redwater, or probably the incubative period of African coast fever is longer than that of Texas fever; and, for one reason or the other, redwater of the usual Colonial type first appeared amongst them, while the "atypical" cases observed later on were probably the result of a predominant infection with African coast fever.

The probability that African coast fever has a longer incubative period than Texas fever, and the occurrence of cases in which the organisms of African coast fever and of ordinary redwater co-exist in the blood of certain animals, offers an explanation for the apparently positive results obtained by certain artificial infection experiments, which seemed to point to ordinary redwater as the sole cause of the present outbreak; as, if blood for inoculation was taken from an animal suffering from two diseases, one of which has a shorter incubative period than the other, the disease with the short incubative period will be the first to declare itself, and, if the reaction produced is one with charac-

teristic peculiarities, the outcome of such an inoculation experiment may be most misleading.

When we began our experiments upon healthy animals we expected to be able to produce the disease without difficulty by subcutaneous inoculation with blood taken from sick animals, an easy matter in ordinary Texas fever, in which the injection of 5 cc. virulent blood almost always induces a severe and frequently a fatal attack; and, as we understood inoculation with virulent blood in African coast fever had had a like effect, our surprise was the greater when we found that such inoculations were ineffective when we injected subcutaneously blood containing an abundance of the small parasites. As it was important to establish beyond dispute that such inoculations do not communicate the disease, many experiments were made with varying doses of blood, and in various ways. Instead of using defibrinated blood as we did at first, warm fresh blood drawn directly from the jugular vein was tried, injections being made subcutaneously, directly into a vein, and into the peritoneal cavity. In some cases the injected blood was mixed with an emulsion of spleen pulp and of lymphatic gland, as these organs contain large numbers of multiplication forms of parasite, as I have mentioned in my first Report. In others virulent blood in large volume was injected, doses of 2000 cc. being introduced subcutaneously, and 500 cc. intravenously, but by none of these methods did we succeed in producing the disease. None of the inoculated animals became sick, nor could the parasites be discovered microscopically in their blood. Even in those cases in which blood containing innumerable parasites was injected directly into the vein we were unable to find a single parasite the following day.

All our experiments, and they were many, indicate that the direct inoculation of healthy susceptible animals with the blood of animals suffering from African coast fever will not reproduce the disease, and this remarkable fact separates still further African coast fever and Texas fever. In the one case no result follows blood inoculation, while inoculation with blood from the less fatal Texas fever is attended by most serious results.

Although such inoculations in African coast fever fail to communicate the disease, they are nevertheless not without effect. Inoculated animals do not become sick, and parasites are absent from their blood, but when these animals are subjected to a second inoculation with virulent blood the result of the second inoculation differs from that of the first, as we have observed that while the first injection of virulent blood produces either an insignificant rise of temperature on the two following days, or no rise at all, after the second inoculation a temperature reaction follows immediately, which persists for one or two days, and which we may look upon as arising from the inoculation; but, what is still more important, after an incubative period of from ten to twelve days, a mild attack of African coast fever supervenes, characterised by the appearance of the usual small parasites in the blood, and generally by a temperature rise of some days' duration. In the course of twenty-one experiments we have succeeded in ten instances in producing a mild attack of the disease, which clearly shows that such a result is not accidental, and we have now to determine which is the most certain method of producing this mild attack, what doses of virulent blood should be given, and what interval should elapse between the doses in order to be certain of inducing such an attack in every instance. Whether the incidence of such a mild attack confers any immunity, I cannot say at present, but a consideration of the effect upon the animal organism produced by mild artificially induced attacks of other infectious diseases leads me to think that some degree of immunity may result.

How great this immunity may be, we will only be able to state when we have discovered a certain method of artificially reproducing the disease in the viru-

lent form which it assumes when arising from veldt infection, and this is another problem which has not yet been solved, although I hope we will overcome this difficulty, either by a modification of some method of direct infection with virulent blood or by working indirectly on the lines in which natural infection occurs, through the medium of the tick.

With this object in view, and for the purpose of ascertaining with certainty what tick may be responsible for the spread of this disease, numerous tick infection experiments have been initiated. Young ticks of all suspected species are being hatched from eggs laid by mature females taken from sick animals, and many have been placed upon healthy susceptible animals, but these experiments have not reached that stage at which we may expect to get conclusive results, tick infection experiments, as I have already remarked, being particularly tedious and taking up much time.

Concurrently with this work, we are conducting experiments with a view to obtaining a curative serum. At first I intended to prepare an antitoxic serum which would tend to neutralise the toxic products of the organism. For such a purpose the animal whose serum it is proposed to use is inoculated with gradually increased doses of virulent blood; and, working on these lines, we have now several animals which are highly fortified, but since we have discovered that healthy animals are also able to resist large doses of virulent blood, I have also decided to prepare animals by inoculation with successive large doses of virulent blood for the purpose of obtaining a cytolytic serum. Such a serum possesses the property of directly attacking the specific parasite, instead of neutralising its products as an antitoxic serum would do, and we have now a number of animals fortified for producing such a cytolytic serum, but more time is required in order to enable us to produce a sufficiently powerful serum of this class for our experiments.

For the production of antitoxic and cytolytic serum only immune animals can be used, as susceptible animals tend to break down under repeated injections of virulent blood, and for our work the immune animals taken over from the Transport Department, Salisbury, which survived the outbreak there, and which have since been grazed continually on infected veldt, have been most valuable. Of their immunity I think there can be little doubt, as none of them have shown any signs of indisposition after repeated injections with large doses of highly infected blood.

Our experience with the animals which we found here on our arrival has been less satisfactory. These animals lent by Bulawayo farmers many months ago had been subjected, previous to our arrival, to a series of inoculations, beginning with recovered and finishing with virulent blood, with a view to conferring immunity on somewhat similar lines to those by which animals are immunised against ordinary Texas fever. Most had been turned out on the Commonage for exposure to natural infection after the final inoculation, and an examination of their blood on our arrival revealed the presence of a few small parasites, which I stated in my first report led me to believe they might be immune, but this unfortunately is not the case, as several (13 out of 29) have since developed acute attacks of African Coast fever and died. From this we must conclude that the discovery of single small parasites in the blood of cattle exposed to infection with African Coast fever only proves that these animals have come in contact with some infecting agency, but does not indicate that they are capable of resisting further infection, as we previously supposed. Herein lies another point of difference between African Coast fever and Texas fever, as the discovery of isolated pyriform organisms in the blood of animals which have been affected with Texas fever can generally be considered to indicate that under ordinary circumstances such animals will resist further infection with this disease, a conclusion unwarranted in dealing with African Coast fever when we detect the presence of isolated organisms in the blood of apparently healthy animals.

The presence of such small parasites in every case in which they are found can be taken, I believe, as a proof that the cattle whose blood contains them certainly come from areas where African Coast fever infection exists. All our investigations on this point bear out such an assumption. Single parasites have been found in the blood of forty animals which have come from the infected areas of Salisbury and Bulawayo, while no such parasites have been found in thirty blood preparations sent from the Cape Peninsula, an area free from both Texas fever and African Coast fever. For the specimens sent from Cape Town I am indebted to Dr Hutcheon, Colonial Veterinary Surgeon, who kindly arranged to have them forwarded to Bulawayo. In the blood of sixty-four animals from the clean district of Plumtree no such single parasites have been demonstrated, nor did an examination of blood preparations of other twenty-four animals, taken in the same district, reveal their presence; while it is particularly interesting to note that in the blood of seven animals brought from Beira, and of the six brought from Dar-es-Salaam (German East Africa) for the purpose of testing their immunity, we found the same small parasites which were present in our local animals.

For the preparation of animals, in order to obtain antitoxic and cytolytic serums, much virulent blood was necessary, and at first its scarcity somewhat retarded our work; but latterly, thanks to the efforts of Mr Marshall Hole, Civil Commissioner, Mr Taylor, Chief Native Commissioner, and Mr Gray, Chief Veterinary Surgeon; to the liberality of certain farmers in the Bulawayo district; and to the energy of Messrs Fynn and M'Donald, in charge of the Fingo Location, Bembesi, who have sent in large numbers of sick cattle, contributed by the Fingoes, we have had no difficulty in carrying out this part of our work.

In all, 78 sick animals have been obtained, which have been of material service, enabling us to carry out a systematic observation of animals while sick, and to obtain virulent blood for fortification purposes while they lived, while such as died furnished valuable material for careful *post-mortem* examinations.

ROYAL VETERINARY COLLEGE, LONDON.

INAUGURATION OF THE WINTER SESSION, 1903-1904.

The winter session of this institution was opened on Thursday, 1st October, when the inaugural address was delivered by Mr J. ASTLEY BLOXAM, F.R.C.S., The large lecture theatre was filled to overflowing, the audience comprising many members of the profession, in addition to Governors of the College, members of the teaching staff, and students.

Mr A. C. COPE, Chief Veterinary Officer to the Board of Agriculture, presided, and, in introducing the lecturer, said that Mr BLOXAM had a two-fold claim on their attention. In the first place, he was a distinguished member of the medical profession, and, in the second place, he was one of the Governors of the Institution.

Mr BLOXAM then delivered the address, as follows:—

"Mr CHAIRMAN AND GENTLEMEN—In glancing at the reports of introductory addresses which have been given here by distinguished members of your body in previous years, I have been much struck with the evidences of progress in veterinary science which they display. It is no part of my intention to attempt to rival these admirable reviews and comprehensive surveys. I am free to confess that my own particular field of work has not brought me, except as an amateur, into contact with your own. But years ago, when assistant-surgeon in the Royal Horse Guards Blue, accompanying the veterinary surgeon on his rounds there, I first learned to appreciate the value of the services which veterinary science

renders not only to suffering animals, but to the cause of humanity at large. And my own humble efforts in animal surgery have tended to strengthen the feeling of admiration I have for the skill which members of your profession exhibit. We seem to be removed a very long way from the days when the horse-doctor on the one hand and the bone setter on the other, enjoyed a more or less enviable reputation. I don't know that either species is quite extinct. The believer in blistering and firing still exists; and perhaps Hutton has still his disciples. But whatever the veterinary was, and whether he was ever all that fancy and the caricaturist have painted him, it is very certain that he has passed out of the transition stage and has become a professional man, with a brass plate and a recognised position, and he deserves it. It gratifies me to think that in general education he is in no way inferior to the average medical student walking our hospitals; that he is probably able to read Pasteur's notes in the original French and Koch's treatises in German, and that he comes to his course of professional study well qualified to pursue it thoroughly and intelligently.

"Well, gentlemen, I think I may congratulate you upon having passed your preliminary examination. You are now entering upon an honoured profession, one which, I am glad to find, has ever met with the sympathy of medical men. Your preliminary examination is high, but not quite so difficult as it is in our profession. May I, perhaps, suggest that the time has nearly come when the standard of admission should be still further raised? It would be, in my opinion, a most advantageous step to make the preliminary examination just a little harder. I am sure you will all agree with me.

YOUR ALMA MATER.

"Now, having joined the College, may I tell you something about your Alma Mater? I speak as a Governor, when I say that the purposes of the foundation have not been altered during the last century. The original and sustained object of the institution is to reform and bring into a regular system that important branch of medicine which regards the treatment of diseases incident to horses and other cattle. It was at Blenheim Coffee House, Bond Street, on 18th February 1791, that the Veterinary College, London, for the reformation and improvement of farriery was founded, and the first professor was M. Vial de St. Bel, who had been for some years Professor of Veterinary Medicine in the Royal School at Lyons and of Comparative Anatomy at Montpellier—"a gentleman well known for his anatomical skill and knowledge in every part of his art." The Duke of Northumberland was the first President. Four acres of land were secured near St. Pancras Church for the College, and a part of the Fleet Ditch formed the College boundary, and, through drains, it was given to inconveniently flooding the premises. There can be no doubt that in those days animals were kept in the pastures adjoining, as the remains of a passage still exist under Great College Street. The animals could be led into the fields, avoiding the streets and the toll-gate.

"I have been privileged to peruse an account of the origin and progress of the College for 1791-1871. It is written by a former principal, Professor James Beart Simonds, who is, I rejoice to know, still alive. He is the author of many essays on veterinary matters, and especially is he famous for his excellent treatises on 'Variola Ovina' and on the 'Age of the Ox, Sheep, and Pig.' These articles have stood the test of time, and their author ranks as one of those men who have done most original work while connected with the College. His efforts in connection with the stamping out of the cattle plague I need not dwell upon. Professor Simond's history of the College, I may say, has some, I hope severely satirical, illustrations, which, if I could throw them upon a screen, would give you an idea of the crude beginnings of the art of horse doctoring, when cropping was performed by a paunchy professor, standing

the Veterinary Surgeons Act secured the monopoly. The teaching equipment of this, your college comprises a staff of Professors and assistants, class rooms, laboratories, museum, library, and hospital accommodation for over one hundred animals. It has also a large out-patient clinique.

"I think I may here say, that whilst it is recognised by the Governors of this, the Royal Veterinary College, that it is wise to prevent competition between a number of degree-granting bodies, which would tend to lower the standard of veterinary education, at the same time, the fact that there is only one qualifying degree or diploma in veterinary science tends to bring the standard to a dead level, and deprives students of exceptional ability of the incentive to further study, which would be afforded by the existence of a degree in veterinary science, generally recognised to be of a higher standard than the one necessary for qualification to practise as a veterinary surgeon. The Governors of this college 'believe that the institution of such a degree by the University of London would do much to stimulate research in matters connected with the physiology and pathology of the domesticated animals, and that it would gradually add to the profession a valuable leaven of highly-trained veterinary surgeons, whose services in the sphere of epizootiology alone would be of immense value to the country.'

"To this end, therefore, at a meeting of the Governors held on 20th July last, Field-Marshal H.R.H. the Duke of Cambridge in the Chair, a resolution was carried unanimously which I will read, for this resolution is now being followed up:—

"'That the Senate of the University of London be approached with the request that the University may be pleased to institute a special Degree in Science, the examinations for this Degree to embrace such subjects as may be considered essential for the higher education and training of scientific veterinary surgeons.'

"Well, gentlemen, you understand that we do not wish to deprive the Royal College of Veterinary Surgeons of the monopoly which it at present possesses of fixing the minimum standard of veterinary education and of conducting the examinations necessary to qualify for membership of the profession. In order to obtain this Diploma, M.R.C.V.S., students must pass, as you have done, the preliminary examination. What follows for students in England? (1) Four sessional years at this College. (2) Four professional examinations.

"Until 1880 it was only a two years' course, and in 1881, three; and it has been since 1893 only that the course has been made four years. The first year is devoted to the study of biology, and includes biology, elementary anatomy, and chemistry. The second year is given to anatomy, physiology, and histology; and there is an examination in these subjects at the end of the second year. The third year is assigned to pathology, materia medica, therapeutics, and hygiene, and at the close another examination. The fourth year is required for medicine, surgery, and meat inspection, with an examination on these subjects. That constitutes the pass. At the end of the second year an examination takes place in stable management, and this I regard as very important, as it is essentially practical.

"To qualify for the Diploma, in short, a student of the Royal Veterinary College must give his attention to nearly 700 lectures, 200 demonstrations, and more than double that number of clinical and tutorial classes. He must attend the examination of 1800 or more animals brought to the College for advice and treatment, and watch the progress of patients in the Infirmary. He must take his share of the practice at the Free Clinique, where 5000 horses and dogs are treated every year. Lastly, he must take an active interest in the examination as to soundness of from ten to eleven hundred horses of all classes.

"All these various lectures and demonstrations, which I hope you may all

of you soon attend, when they are given by your Professors, are not only necessary to secure the Diploma, but they are intended to help you to attain the art of teaching yourselves, and that of forming your own conclusions in the practice of your profession. And you are to remember from this day forward that to be successful veterinary surgeons you must make it your everyday study to observe domestic animals, not only in disease, but especially in health.

"I am tempted, perhaps, to consider that one day greater attention will be paid by your examiners to that part of your studies which relates particularly to surgery.

"Many things are now done to secure the advantage of aseptic surgery in your operating rooms. I know of the very great difficulties you have to encounter. I know, too, that animal life is held comparatively cheap. In mankind it is otherwise, and human life is preserved though the body be reduced to a trunk. Obviously a horse minus a limb is useless. But there are many times when it is most desirable that the operation should succeed, or why undertake it? all and aseptic surgery vastly increases the probabilities of success. I know that what we in human surgery call major operations frequently are undertaken. I heard the other day of a poor suffering dog with contracted pelvis being admitted and having a cæsarean operation performed, with removal of the uterus and appendages by your resident house surgeon successfully. Think what a joy it must have been to the master, and what pleasure it must have been to the operator to know that his efforts had been rewarded. In these operations, and in fact in all, let me impress upon you the importance of giving anæsthetics. Not only do you by these means banish time as a factor, but they render operative action easier and you relieve poor suffering animals from pain, whilst nerving your own hand to do its delicate task.

"I agree that failure may not follow the use of primitive tools. I recollect that I operated once upon a cow for tumour, and the only means I had to arrest hæmorrhage was a packing needle and a piece of string. The animal was wasting away when I saw it, walking about with a seton in the great lump, and there was suppuration. I said to the farmer, 'I will remove it if you will get me a waterproof coat.' And with a gardener's knife I removed the tumour—it was a fatty cystic tumour and it weighed twenty pounds. I directed a man to syringe the wound with carbolic lotion, and the cow recovered perfectly and was sold for a good sum afterwards. I fear I acted unprofessionally, but I hope I may be forgiven by the profession.

"This incident and others of the kind, with the occasional treatment of a broken leg of a dog to oblige its fair owner, have taught me that we must not lose sight of the fact of the great difficulty that you in your profession are compelled to face because animals are not able to articulate their feelings; to tell us what they would wish us to know and which would be so great a guide. It is in this respect that the veterinary surgeon works at a manifest disadvantage, and I think that it is incumbent upon him to be most diligent in observing symptoms, even the slightest evidences of illness, which would enlighten him as to the cause of the evil.

"You have, too, a greater difficulty in administering medicines, and in giving nutriment to sick animals than we have in treating human beings, and may there not be a field of inquiry as to the possibility of nourishing such animals in some better way than you even now possess.

"Many problems await solution in which I think the veterinary profession can help us in the treatment of human diseases. Take, for example, what we call cancer and its allied growths. Why are certain new growths, which much resemble cancer, more common in grey horses? Why do new growths, resembling sarcoma, occur in dogs after injuries? We also know now by your help that certain new growths are directly contagious. All these are

matters of importance, and who can say but by your help somebody may arrive at the cause of these terrible diseases, and hence probably produce a cure? Every fact of importance in these days is of extreme value, and must help those who are now specially engaged in the investigation of cancer in the human subject.

HOUSING OF ANIMALS.

"To my mind there is a large field of inquiry, not only with respect to the purity of the milk supply, but the whole of the sanitary conditions in which cows, cattle, and horses are kept. Between the animal kingdom and mankind there is the closest possible connection, and we shall be wiser when we pay a greater degree of attention to sanitation, enforced by a system of inspection, which at present, if it exists at all, exists in a very limited degree.

"It should be the business of local authorities to see that animals are properly housed. We are too prone to lock the stable door after the steed has been stolen, and to decry the expense and interference of what has been styled paternal administration. The public interest, in my opinion, demands that an end should be put to the foul, evil-smelling, excreta-laden, rank, neglected farm-yards, the tumble down cow-sheds, the wretched pig-styes, which are a disgrace to any kind of farming, high or low. Are not cattle sometimes grazed year after year in the same meadows and pastures without change of venue at all? Is it not possible to imagine that in antiquated buildings and exhausted land there may lurk some of the undiscovered contagia of diseases whose origin have defied every research and whose recrudescence is a mystery? For instance, some years ago Mr A. C. Cope, the Chief Veterinary Officer of the Board of Agriculture, tells us that when foot-and-mouth disease was very rife in the Deptford Cattle Market, and at a time when the United States was free from disease, some cargoes of cattle were found to be affected upon their arrival at the ports of Liverpool and London from America. The matter was made the subject of inquiry, and for a long time without success; but it subsequently transpired that it had been the practice on the part of the men in charge of the cattle imported to take back to the States the head-ropes which had been kept for some days in Deptford market, and to use them again for other consignments on their next journey. It was therefore surmised that these head-ropes had become infected at Deptford, and had thus been the means of conveying the disease to the healthy cattle brought in succeeding vessels. The surmise proved to be correct, because immediately the practice was abandoned and new ropes supplied each succeeding cargo arrived healthy.

"Now, if head-ropes can be a source of danger, why should not sheds and barns be equally so? When foot-and-mouth disease reappeared in Great Britain last March twelvemonth, we had enjoyed freedom from that disease for nearly one year. Searching investigations were made with a view to discover the origin of the outbreak, but without eliciting any definite clue as to the source of the contagion. The disease was, however, at that period prevalent in Western Europe. Again in August last there was the mysterious outbreak in America, and its origin has not been accounted for, but I think the Americans took the right course in stamping it out by wholesale slaughter of the diseased and exposed animals, and disinfecting the buildings in which they had been sheltered. I am even disposed to go further in thinking that infected premises should also be destroyed until we know something more of the conditions in which diseases are spread. For it appears to me that we are only at the beginning of our researches into the subject of contagium, and that veterinary students cannot devote too much time to following up the inquiries of Pollender, Pasteur, and Koch. Whilst we know what causes anthrax and glanders, and the contagium of swine fever has been determined by bacterio-

logical research, the contagia of cattle plague, foot-and-mouth disease, and others, all remain to be discovered. It is true we know enough of the nature of some to have combated these diseases with astonishing success.

"It is more than a quarter of a century since we had a visitation of cattle plague in this country. This was in 1877 and was the third, the first outbreak in 1865-67 having found the country entirely unprepared with any kind of veterinary organisation, and as a result not less than 400,000 cattle died or were slaughtered before the disease which spread all over Great Britain was eradicated. The second outbreak in 1872 was under the Veterinary Department of the Privy Council, and was promptly stamped out, as also was the third attack.

FOOT-AND-MOUTH DISEASE.

"Foot-and-mouth disease from 1887 to 1891, and again from 1895 to 1899, was non-existent, but it is a disease against which we must be continuously on our guard, and no one, I think, will question the wisdom of stopping the importation of live cattle from Argentina, so long as any danger from its reintroduction exists. Our cattle population does not grow proportionately with our human population. We have fewer beasts now than we had four years ago, the total scarcely exceeding six millions and a half, which includes the cows that furnish our milk supply. Of sheep, we have a million less; and swine, too, are declining in numbers. Of course we should soon increase the totals if we were to extend to Canada the privileges enjoyed by Ireland, the sister country sending us of fat and store cattle and calves nearly one million animals yearly, besides another million sheep, and over six hundred thousand swine. But although it is a vexed question, I do not anticipate that Canada will, notwithstanding that the Dominion is remarkably free of disease, secure the relaxation of the restrictions, for the reason that Canada is not close at our doors, and we should be unable to keep such a strict eye upon our imports as we are enabled to do by means of veterinary inspectors upon shipments of animals from Ireland.

RABIES.

"I believe that it is now fully a year since rabies was reported in this country. Last October, or thereabouts, the Board of Agriculture had the end of a stubborn outbreak to deal with in South Wales, and about the same time there was a case in Cornwall. It would be, perhaps, too much to say that the disease has been stamped out for good and aye, for we have had these periods of fancied security recorded previously and yet have discovered that we were mistaken, and no one who has witnessed the horrors of hydrophobia can for a moment say that any sacrifice is too great to guard us against its recurrence. Muzzling orders may never be popular, but they are undeniably the means which have been found uniformly successful in combating an outbreak. I believe that the Board of Agriculture will, whenever necessary, put rigorously into force its powers, through the local authorities, to muzzle all dogs in public places sufficiently long to allow of the development of latent disease in animals which might have been in contact with a rabid dog. In South Wales restrictions of a very stringent nature became necessary, following upon the outbreak which lasted practically the first nine months of 1902. It was traceable to a stray dog which had the appearance of having travelled a considerable distance, and before it was killed it had bitten two children, several dogs, and other animals. Now, rabies had declared itself five months after the rabid animal was itself bitten. Five months, I believe, is nearly the longest interval recorded between the bite and the manifestation of the disease in a dog. In man the interval is sometimes much longer. Certain it is that on 28th May 1902 an unfortunate man died of hydrophobia at Garnet, Carmarthenshire. He had been bitten two years previously in the same neighbourhood by a dog which was not at the time recognised as rabid.

"May I not, therefore, draw from this incident the moral, that with the greater infrequency of rabies, and, as I may hope, its total extinction, veterinary students should not treat the disease as no longer concerning them, but should pay the greatest attention to its symptoms, in order to be able to recognise it when confronted with a suspicious case?"

"The services of the veterinary surgeon are in demand whenever dogs are imported. Owners are no longer at liberty to isolate the imported dog themselves for the required period of six calendar months after landing. The dog must be detained upon premises in the occupation of or under the control of a veterinary surgeon, which the Board of Agriculture has approved as a place of detention. Any premises in the occupation of a veterinary surgeon in Great Britain may be proposed to the Board of Agriculture, but approval rests with the Board. Most of the foreign dogs imported into London, I am told, are taken to a Sanatorium near Croyden, managed by a veterinary surgeon. Strict isolation is insisted upon, even when at exercise, and the dimensions and construction of the kennel are such as enable the dog, regard being had to its breed and size, to be kept in comfort and health. No dog can come into contact with another dog during exercise in a suitable enclosure. A weekly return as to the health of all imported dogs has to be furnished to the Board by the veterinary surgeon.

"As to the effect of the amended regulations, which are, of course, designed to afford security against the re-introduction of rabies, and therefore ought not to have been the occasion of friction, the importations are, I believe, now diminishing. Last year the close of the war in South Africa, with the consequent return of any number of regimental pets—by no means all of the canine species—led to no diminution. That abnormal importation has now ceased. There is still an element of danger, reduced though it has been by the vigorous enforcement of the order by the officers of Customs—I refer to the carrying of dogs on vessels trading with foreign ports. As they are not landed officially, they escape isolation; but who is to prevent a dog which is supposed to stay on board during the time the vessel is in dock from making an unofficial peregrination of the neighbourhood? And if he is infected with rabies, what becomes of all our attempts to stamp out the disease?"

"It was for many years believed that the contagium of rabies existed only in the saliva of the affected animal, but the late M. Pasteur experimentally demonstrated that it is also present in the structure of the brain and spinal cord, and that by a special method of drying the cord the virus of the disease becomes attenuated, and when thus prepared can be used as an antitoxin for hydrophobia in man. Personally I much prefer the policy of prevention to that of cure. A muzzled dog has my sympathy, but the muzzling of our canine friends is better than canine madness in humanity.

TUBERCULOSIS.

"Quite recently a great congress of medical and scientific men at Brussels again considered the remarkable theory of Dr Koch, that bovine tuberculosis is not transmissible to human beings. Dr de Jong, Professor at Leyden University, believes that it is transmissible. Human tuberculosis, he pointed out, had many times been communicated to cattle by means of a general infection. The bacilli of human and bovine tuberculosis were, he argued, identical in their effects. Dr Gratia, of Brussels, also contended that proof had now been given that human tuberculosis was transmissible to cattle. It had been maintained that proof was not complete because the infection was only local. The converse experiment (the transmission of bovine tuberculosis to man) could not be made. Dr Gratia, however, declared that he had performed a similar experiment on sixteen monkeys by giving them the milk of tuberculous cows to drink, and fourteen of the animals died. He also reported other experiments which had been made with regard to infantile

tuberculosis. Sixteen per cent. of the children examined had been found to be tuberculous through being fed with milk from tuberculous cows. That, too, showed that bovine tuberculosis was transmissible to man.

"M. Kessel, a pupil of Dr Koch, maintained that the inoculation of cattle with human tuberculosis had only produced local results from which no conclusion could properly be drawn.

"The discussion of the Koch theory proved so animated that the sitting lasted over five hours. The debate resolved itself largely into a controversy between the French and Belgian delegates on the one hand and the Germans on the other. The former supported the thesis of the transmissibility of bovine tuberculosis to human beings, the speakers to this effect including MM. Chauveau, Gratia, Halloin, and Mentanas. The Germans—notably Dr Kirsch—contended that such transmissibility had not been proved.

"Eventually the sections by a large majority passed the following resolution:—

"That human tuberculosis is perfectly transmissible from one person to another. Nevertheless, in the present state of our knowledge, it is necessary to recommend hygienic measures for the prevention of the propagation of animal tuberculosis to the human species."

"That I take to be a remarkable contribution to the interesting controversy which is still keen. We are awaiting the results of inquiry. Dr Koch's discovery of the bacillus of tuberculosis has proved that the disease is produced by contagium and not mainly due to heredity. The newer theory leads us to entertain the hope that consumption in man and beast may be after all curable, and yield to the application of rigid hygienic principles.

"It is clear that the same conditions apply to both animals and human beings in the production of tuberculosis, and its prevention can only be met by strict attention to hygiene. In animals its presence can always be proved by tuberculin injection, and the animal killed at once. It is better in such a case to kill. But is this practicable, having regard to the enormous number of animals that are tuberculous? and the same observation applies to glandered horses. It is a question of monetary loss. Nevertheless, it would be better to kill than attempt to cure. Mankind will not submit to extermination for the benefit of the race, and asks us for a cure, and we have still to find it.

IN CONCLUSION.

"When in these days so much attention is being given, as we have seen, to tuberculosis in man and beast, and the subject of cancer research is all engrossing, may I not suggest that the Government should recognise the importance of the services of veterinary science to agriculture and to mankind in general by making a grant in aid of veterinary education and research, or if possible establishing a Convalescent Home or Farm for animals, a place where sound animals could be studied. There is a precedent for such a grant, as I believe £1500 was annually paid by the Government towards the maintenance of this College until the early part of the nineteenth century, and recently the Irish Veterinary College has received £25,000.

"No doubt the profession has made great advances during the past century, and more particularly during the last twenty years, and the veterinary surgeons who have been educated in this and other veterinary colleges have rendered great service to the country, notably in dealing with the contagious diseases of animals. There can hardly be a doubt, however, that if veterinary education is to march with the times, and if this country is to bear its part in the advancement of veterinary knowledge in the future, the State must follow the example set by other countries, and contribute handsomely to the equipment and upkeep of the veterinary schools."

The CHAIRMAN, in proposing a vote of thanks to the lecturer, said that they

had had the pleasure of listening to one of the most interesting addresses which had ever been delivered from that table. Mr Bloxam had dealt with the education of the students of the profession, and had told them of the steps which had recently been taken with the object of inducing the University of London to institute a higher degree in veterinary science. Further than that, he had given them a history which astounded him from its accuracy, and led him to believe that Mr Bloxam must have studied all the reports that had ever been written by the Board of Agriculture, not only with regard to the nature of the diseases of animals, but also concerning the methods which had been employed to stamp them out. They were under a great obligation to Mr Bloxam for having given them such an excellent address, and he proposed that they accord him a very hearty vote of thanks.

The vote of thanks was carried with acclamation, and Mr BLOXAM, in acknowledging the compliment, said that it had been a great pleasure to him to deliver the address. He felt that he was personally indebted to their profession for many facts which had been of use to him in the treatment of members of his own race. He was convinced that they (the students) would in the future help in solving many great problems, and in the discovery of many important facts which would tend towards the prevention of disease among human beings.

The CHAIRMAN said it occurred to him that he ought to take the opportunity of expressing their regret at the loss which the College had sustained since the close of the past session through the retirement of two members of the staff, viz., Dr Power and Mr Bayne, who had rendered good service to the institution and to the profession by the excellence of their teaching. At the same time he desired to offer a word of welcome to their successors, Dr Brodie and Dr Lander. These gentlemen joined the staff of the College with the very highest recommendations as to their abilities, and he did not doubt that they would do their utmost to impart the knowledge which they possessed to their students.

On the motion of SIR GEORGE BROWN, a hearty vote of thanks was accorded to the CHAIRMAN, and the proceedings then terminated.

ROYAL VETERINARY COLLEGE.

LIST OF BURSARIES, MEDALS, CLASS PRIZES, ETC., 1902-1903.

Coleman Prizes.

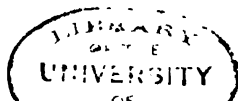
<i>Silver Medal</i>	Mr E. O'Reilly.
<i>Bronze Medal</i>	Mr H. L. Jones.
<i>Certificate of Merit</i>	Mr F. C. O'Rorke.

Centenary Prizes (£20 each).

<i>Class A</i>	Mr A. L. Sheather.
<i>Class B</i>	Mr L. Danels.
<i>Class C</i>	Mr E. Brown.
<i>Class D</i>	Mr H. L. Jones.

Royal Agricultural Society's Medals.

<i>Silver Medal</i>	Mr T. Bowett.
<i>Bronze Medal</i>	Mr H. L. Jones.



Class B.

ANATOMY.—*First Prize*—L. Danels. *Second Prize*—W. T. M. Browne. *Second-class Honour Certificates*—J. Godber, W. H. Thomas, W. H. Taylor.

HISTOLOGY.—*First Prize*—J. Godber. *Second Prize*—L. Danels. *Second-class Honour Certificates*—R. Branford, W. T. Brookes, H. A. Lake, D. C. Matheson, G. C. H. Sharpe, W. H. Thomas, W. H. Taylor.

PHYSIOLOGY.—*First Prize*—J. Godber. *Second Prize*—L. Danels. *Second-class Honour Certificates*—W. A. Broad, R. Branford, E. Belcher, F. Chamberlain, H. A. Lake, D. C. Matheson, W. H. Thomas, W. H. Taylor.

Class A.

CHEMISTRY AND TOXICOLOGY.—*First Prize*—H. R. Singleton. *Second Prize*—A. L. Sheather. *First-class Honour Certificates*—E. R. Blamey, L. B. Cole, M. Henry, C. Hartley, M. H. Kingcome, H. Morphew, A. W. N. Pillers, S. Smith, F. Ware. *Second-class Honour Certificates*—W. E. Blackwell, H. Bone, J. R. Crane, P. L. Edward, M. C. Foreman, F. B. Greer, E. V. Hobbs, F. E. Jones, B. A. Jarvis, P. J. Kerr, V. C. Leckie, T. Lishman, G. T. Matthews, J. R. V. Nasmyth-Miller, V. Pride-Jones, C. H. Strachan, A. E. Webber, W. R. B. Wakeham.

PRACTICAL CHEMISTRY.—*First Prize*—L. B. Cole, B. A. Jarvis (æq). *Second Prize*—E. D. Sewell.

BIOLOGY.—*First Prize*—A. L. Sheather. *Second Prize*—H. Morphew. *First-class Honour Certificates*—M. Henry, M. H. Kingcome, H. R. Singleton. *Second-class Honour Certificates*—W. P. B. Beal, W. E. Blackwell, E. R. Blamey, L. B. Cole, C. Hartley, E. V. Hobbs, F. E. Jones, B. A. Jarvis, V. C. Leckie, T. Lishman, A. W. N. Pillers, C. H. Strachan, S. Smith, A. E. Webber, W. R. Wakeham, F. Ware.

MINOR ANATOMY.—*First Prize*—M. Henry. *Second Prize*—M. H. Kingcome. *First-class Honour Certificates*—H. Morphew, A. L. Sheather. *Second-class Honour Certificates*—W. E. Blackwell, E. R. Blamey, G. H. Carter, J. R. Crane, C. Hartley, E. V. Hobbs, F. E. Jones, B. A. Jarvis, P. J. Kerr, T. Lishman, J. R. V. Nasmyth-Miller, A. W. N. Pillers, C. H. Strachan, S. Smith, H. R. Singleton, W. R. B. Wakeham, F. Ware.

PASS LIST.

The following are the Pass Lists¹ of this Institution for Session 1902-1903.

FIRST PROFESSIONAL EXAMINATION.

Messrs A. K. B. Byles, W. G. Blackwell, L. S. Balls, *A. G. Doherty, R. L. L. Hart, *J. H. Jones, J. H. Jarvis, *R. W. Mellard, *E. R. Nichols, G. B. C. Rees-Mogg, G. C. H. Sharpe, D. Anderson, C. S. Addison, W. P. B. Beal, W. E. Blackwell, E. R. Blamey, H. Bone, J. R. Crane, P. L. Edwards, F. B. Greer, *M. Henry, *C. Hartley, E. V. Hobbs, F. E. Jones, P. J. Kerr, †M. H. Kingcome, P. G. Ledger, *V. C. Leckie, T. Lishman, *H. Morphew, G. T. Matthews, R. C. Moore, †A. W. N. Pillers, E. D. Sewell, C. H. Strachan, *S. Smith, *H. R. Singleton, *A. L. Sheather, A. E. Webber, W. R. B. Wakeham, *F. Ware.

SECOND PROFESSIONAL EXAMINATION.

Messrs A. C. Anderson, R. S. Audas, P. T. Bolton, J. A. Bosley, L. L. Dixon, E. G. Haskell, E. S. Martin, H. Platt, J. M. Richardson, S. Speer, W. K. Townson, J. O. Andrews, W. A. Broad, W. T. M. Browne, R. Branford, *E. Belcher, W. T. Brookes, T. A. Blake, F. W. Chamberlain, A. Chisholm,

¹ In this and the succeeding Pass Lists † indicates with First-Class Honours, and * with Second-Class Honours.

L. Danels, J. Forrest, J. Godber, B. M. Gunn, T. Hicks, J. R. Hewer, A. L. Horner, H. A. Lake, T. R. Lydford, A. Munro, B. McGuire, D. C. Matheson, S. H. Nixon, F. T. Prince, *R. B. Palmer, *C. C. Parsons, C. Pinchin, C. S. Simpson, G. G. Sooby, *W. H. Simpson, W. H. Taylor, W. H. Thomas, W. D. Williams, *H. S. Ward, A Young.

THIRD PROFESSIONAL EXAMINATION.

Messrs T. E. Burridge, E. T. Clemow, J. McRae Frost, *R. C. Glover, J. Hobday, J. T. M. Hughes, *W. G. Litt, T. A. Nicholas, *H. K. Roberts, J. Richardson, D. O. Turnbull, H. M. Williams, J. J. Aveston, *R. Bennett, J. Blakeway, *L. E. W. Bevan, *E. Brown, T. Bone, R. Casey, W. T. Collins, H. C. Dibben, W. J. Dale, †T. C. Graves, *J. R. Hodgkins, B. H. Lane, *W. J. Moody, G. O. Ogden, †R. L. Phillips, D. Quinlan, H. W. Quinlan, H. Sproston, F. V. Steward, L. S. Sedgwick, W. Trigger, R. Tindle, *W. Urquhart, E. R. H. Woodcock.

FINAL EXAMINATION.

Messrs J. G. Bush, A. Cornish-Bowden, N. Gilford, J. C. Hally, F. T. Hancock, R. E. Klyne, A. C. Lloyd, W. P. Loft, W. L. Little, A. J. MacDonald, G. E. Owen, F. O. Parsons, P. G. Short, C. H. Sheather, A. V. W. Sewell, F. H. Sugden, A. J. Thompson, W. F. Wilson, T. Bowett, W. Cawthorn, D. R. Davis, G. A. Dibben, A. N. Foster, W. S. Gillespie, H. J. Holness, R. J. Hills, H. L. Jones, A. S. Leese, H. P. Lewis, H. Mason, F. D. Neal, E. O'Reilly, F. C. O'Rorke, J. H. Ripley, W. A. Simson, V. de V. H. Woodley.

DICK VETERINARY COLLEGE, EDINBURGH.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1902-03.

FIRST PROFESSIONAL EXAMINATION.

Messrs T. N. Critchley, J. A. Gault, C. L. O'Gorman, G. W. Rutherford, L. L. Steele, J. W. Sugden, *W. J. Young, J. Anderson, H. Andrew, J. Andrew, F. V. Bagshaw, C. Berry, H. Gibson, H. D. Gilmore, *G. H. Jelbart, G. C. Inglis, J. A. M'Lachlan, *A. M'Turk, F. W. Medlock, T. Thomson, C. E. Waddy, W. R. Watson, W. White.

SECOND PROFESSIONAL EXAMINATION.

Messrs J. M'Clure Barry, F. Drinkwater, J. R. Hayhurst, O. Hatch, *A. E. Massey, W. F. M'Connell, *C. W. Cartwright, C. R. Chadwick, F. Davidson, W. Laing, J. H. M'Crea, J. T. Muirhead, *W. L. Richardson, B. Selous, *H. W. Stevens, H. A. Stewart, A. Taylor, G. Taylor, R. W. Thomson, F. S. Warburton, *A. E. Willett.

THIRD PROFESSIONAL EXAMINATION.

Messrs G. W. Davidson, A. J. Horner, J. Henderson, J. O. Powley, J. S. Bowden, J. Craig, G. Crowhurst, A. de R. Gordon, A. B. Howe, T. T. Jack, *D. S. Rabagliati.

FINAL EXAMINATION.

Messrs R. U. Lewis, W. Robertson, J. W. D. Serjeant, J. D. Whitty, W. K. Barron, *R. E. Montgomery, R. Murray, H. Yeoman.

GLASGOW VETERINARY COLLEGE.**PASS LIST.**

The following are the Pass Lists of this Institution for Session 1902-03.

FIRST PROFESSIONAL EXAMINATION.

Messrs *J. Lawther, P. M'Intyre, A. M'Crum, J. H. Thomson, *W. T. Lindsay, A. C. Perkins, W. Reid, H. H. Brodie, V. M'Leish, R. Dickie, D. B. Rodger.

SECOND PROFESSIONAL EXAMINATION.

Messrs J. Hill, A. Douglas, W. Anderson, G. R. M'Call.

THIRD PROFESSIONAL EXAMINATION.

Messrs H. M'Intyre, K. M'Kenzie, J. Tait, W. Gardner, R. Bickerton, J. B. Thorn, W. W. Campbell, J. Spreull, W. P. Begg, D. M'Leod.

FINAL EXAMINATION.

Messrs J. Crook, J. M'Farlane, J. Wallace, F. Timiny, S. S. Woodrow, J. F. Rankin

NEW VETERINARY COLLEGE, EDINBURGH.**PASS LIST.**

The following are the Pass Lists of this Institution for Session 1902-03.

FIRST PROFESSIONAL EXAMINATION.

Messrs *G. C. Cochrane, *W. Walker, J. G. Runciman, R. G. Wood, O. Carlyle, G. Gordon, E. Greenway, G. M. Hallen, F. M'Donald, J. H. Primmer, J. Smith, J. Taylor, *P. Thexton.

SECOND PROFESSIONAL EXAMINATION.

Messrs C. Elphick, G. H. Petch, W. G. Darling, R. C. Allinson, R. F. Bett, W. Catterall, J. Sheffield, C. Taylor.

THIRD PROFESSIONAL EXAMINATION.

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ROYAL VETERINARY COLLEGE OF IRELAND.

PASS LIST.

The following are the Pass Lists of the Examination for Session 1906-7.

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*D. Fitzgerald, T. J. Ryan, E. Young, A. C. Telford.

SECOND PROFESSIONAL EXAMINATION.

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THIRD PROFESSIONAL EXAMINATION.

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FINAL EXAMINATION.

Mr S. Craig.

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**THE WEAR AND TEAR OF HORSES DURING THE
SOUTH AFRICA WAR.**

By Lieut. A. S. HEAD, A.V.D., Pretoria.

THOUGH the war in South Africa was essentially a "horse war," very little of a purely veterinary nature has yet been published regarding it. It is possible that the statistics of the war will have to be limited to the probable number which died, were abandoned, or destroyed, and the figures will mainly have to be arrived at indirectly, rather than by actual detailed accounts.

In war it is difficult to keep returns or records, and even when made they may be lost any day. The careful record of months may be the first thing to be carried down stream if any difficulty occurs in crossing a river. Further, there are often matters more urgent to attend to than the compiling of a list showing what has become of the horses belonging to a regiment.

The question of wear and tear amongst horses is one in which I have felt deeply interested, and from the time when I began this campaign I determined to keep accurate records of disease and mortality. I offer the figures obtained in the form of three tables. They refer to one regiment of cavalry, and cover the period November 1899 to June 1902.

Such details as I have to give are more in the form of a diary in which is noted the length of the marches, the sickness, daily losses, and any other points of interest.

The regiment to which I was attached landed at Capetown in November 1899 with 406 horses in fair condition, in spite of the fact that they had had a bad sea voyage and had just recovered from mange.

After a few days rest at Capetown they entrained to the Colesburg district, about 600 miles away, where the regiment remained three months. At this place mange again broke out, but isolation and treatment stopped the outbreak, though a fresh case would occur every now and then.

The military operations for these three months were practically defensive ones (holding a long line of country), so that the work done by the horses was not great, consisting mainly of reconnaissances, scouting, etc. In consequence, the horses kept in fairly good condition; in fact, out of the sixty that died or were destroyed during that time, thirty-one were killed in action, and twenty were destroyed for bullet wounds (incurable). Fighting went on more or less every day, but nothing on a big scale.

In February 1900 one squadron, consisting of ninety-four men and horses, left with General French to relieve Kimberley, marching 80 miles in three days. They lost five horses from bullet wounds, and had ten sick at the end of the march, of which six were cases of sore backs. This must be considered a good performance, and shows what fit horses are capable of doing.

At the time of this march horses carried a heavy regimental saddle, sword, carbine, full wallets, picketing peg and rope, the man's blanket and cloak, a horse-shoe case with two shoes in it, 20 to 24 lbs. of oats, and two days rations for the man. The total weight of this is nearly 9 stone, and, when the man mounted with his lance and 150 rounds of ammunition, the horse could not have carried much less than 20 stone. During the relief march it was hot in the daytime, and the horses were saddled up before daybreak, and rarely had them taken off before dark.

When the above is taken into consideration with the small number of casualties that occurred in this march, one cannot fail to be impressed with the value of condition, for all these horses were fit.

The Kimberley squadron then moved in the direction of Bloemfontein, taking part in the battle of Paardeberg, and arriving at Bloemfontein on 13th March 1900. They lost thirteen horses from exhaustion on the march, a distance of 100 miles, and arrived with twenty sick, of which twelve were suffering from sore backs.

The two squadrons remaining in the Colesburg district left there on the 6th March 1900 with 297 horses, and marched by easy stages, unopposed, through the country to Bloemfontein, a distance of 200 miles, arriving there on the 17th April 1900. Although the actual marching was not severe, the cavalry took part in some very long reconnaissances, one day doing 47 miles. Their casualties up to Bloemfontein were three horses destroyed for exhaustion, one died, and twenty-two sick at the end of the march, of which ten had sore backs.

At Bloemfontein we rested until the 6th May 1900, and received remounts making our strength up to 447 horses. The remounts were issued only a few days before we marched, and had either just arrived at Bloemfontein by train, or had been trekked up from the Orange River, a distance of 150 miles. A good many of them were only half-broken Argentine ponies, recently shipped to South Africa, and fed during the voyage on hay, probably never having seen oats until issued to the troops. They were taken at once on long marches, with no

time for grazing, and given oats which they were not used to and would not eat. Their condition was soft, and, having to carry 20 stone, it was not surprising that they died of exhaustion and got sore backs.

The regiment with General French's force marched to Kroonstad, a distance of 120 miles, taking part in the battle of Sand River, in which it lost thirty-four horses killed in action.

It is wonderful how well Mauser-bullet wounds do if not interfered with. I have had horses shot through the bones of the leg, the abdomen, and the lungs, and in the great majority of cases they were able to be led along with the troops, and quite fit again in two weeks. The bullet wound seals itself at once with coagulated blood, and heals without the formation of any matter, and without becoming septic if the blood clot is not washed or picked off. This is very important in a country of dust and flies. All a bullet wound really requires is a little iodoform dusted over it to keep off the flies, and not to convert a simple wound into a serious one by attempting extraction.

These remarks about bullet wounds necessarily only apply to those cases attended to in the field. My table shows that 65 per cent. of bullet wounds either cause death outright, or so seriously injure the animal that it cannot leave the field. In a small percentage of cases animals which can carry their rider out of action were found subsequently to be incurable, and are shown in the column "destroyed." The horses with bullet wounds that accompanied us on the march were mainly those in which the wounds implicated the soft structures, and it is to these that my remarks about non-interference apply.

The casualties from Sand River to Kroonstad were, fourteen horses died from wounds and exhaustion, two had to be abandoned, and sixty-three remained sick at the end of the march; of these, twenty-eight had sore backs, and the majority of the remainder were debility cases and exhaustion, eleven of which were sent to the veterinary hospital.

During the period just described the day's march consisted of 20 miles from point to point, rarely going out of a walking pace the whole day, and with many halts. We seldom saw the place we camped in, as we always left before daybreak and camped after dark. The horses were watered and fed before leaving camp in the morning, when it was very cold, and many of them would not drink. They were not watered again until mid-day, when they received their mid day feed without being off-saddled, and on coming into camp in the evening they were again watered and fed. The water was nearly always muddy, it often had dead animals in it, and, after the first few horses had been in it to drink and stirred the mud up, it was as thick as soup, so that a horse that was particular about its water would not drink.

Two and sometimes three days' forage (20 and 30 lbs.) of oats were always carried on the saddle in nose bags. If the horse had received all the forage he was supposed to have, it would not have been enough for one doing the work and carrying the weight, but, as a matter of fact, the nose bags often leaked, and the animals went extremely short. A thoroughly acclimatised horse could not have stood this treatment, and when one considers the raw soft remounts, just off board ship, that were supplied, the mortality is not surprising.

Death or sickness from any other cause than exhaustion and sore backs was very rare.

At Kroonstad the regiment rested for a week, and for the first three days the horses lived on what could be collected in the surrounding country, as the supplies had not come up. We fed the horses on mealies, oat straw, green mealie stalks, wheat, and kaffir corn. The only precaution we took was not to give them too much at a time, and to be careful about their watering. I never saw a case of colic or any bad results from this feeding. All the horses of the regiment were in a very bad way—thin, tired, miserable-looking objects, with sunken eyes, drooping heads, and nothing but skin and bones. A few were so weak that when they got down they had to be helped up in the morning. They did not seem to improve at all with the rest; in fact, they were mostly too far gone, and nothing short of six months rest with soft food would have done them much good.

The march on Pretoria *via* Johannesburg was begun on the 20th May 1900, a distance of 175 miles, and we arrived there on the 6th June 1900. On this march the horses suffered from cold at nights (there was a layer of ice on the water in the early morning). They had no rugs, were in very poor condition, and the cold nights were a great contrast to the hot days. There was no grazing, as the Boers had burnt all the grass behind them.

Although the day's march was not heavy, the horses in the wretched state they were in could hardly do it at a walk; ten died, twenty were destroyed, and seventy had to be abandoned (with their saddlery), as they were too exhausted to go further. Abandoned horses invariably died, as they were too exhausted to get better; they ended by getting down, and, being too weak to rise, lay there and died in one or two days without food or water. Thousands died when they had the best food and attention in a veterinary hospital, as they were in such an emaciated and weak state that recovery seemed quite impossible.

There were three days' fighting before Johannesburg, but without casualties amongst the horses.

On arrival at Pretoria there were seventy-two sick horses, of which thirty were suffering from sore backs; and 196 horses, the remnant of the regiment, still working but in a very weak and exhausted state. This remnant shared in the battle of Diamond Hill a few days later, but luckily the horses had not a heavy time, for at this period they were most miserable-looking objects of skin and bone, with sunken eyes and hanging heads, just able to walk. *This was Cavalry in war!*

After the battle of Diamond Hill the regiment went into camp and was re-equipped and remounted up to 556 horses, mostly of the Hungarian breed.

Hungarians are thoroughly useless horses. In my two years and six months trekking not 10 per cent. of all the Hungarian horses issued to the regiment have lasted to do any work. They are mostly weak, flat-sided, light-boned, round-jointed, long-bodied animals, which look pretty when fat and sleek, but tuck up and go to pieces with one day's work—in fact, they are what are called "flat catchers."

At this time the saddles issued to the regiment were called "the American Army Regulation Saddle," I believe—a sort of bad Mexican saddle, with no buckle for the girths, but fastened with a raw hide

rope, knotted through a loop on the saddle. The saddle consisted of two side boards, a front and hind arch, and a seat open over the spine, under which a blanket was placed. These saddles were all handed in again, as they were a failure, and Colonial saddles drawn, which I think are far and away the best saddle for a campaign, as they can easily be made to fit any horses back on the march, and they are light.

The regiment left Pretoria on the 9th July 1900 with 556 horses, and in two days marched to within a few miles of Springs, a distance of 40 miles. It was now that the uselessness of the Hungarian horses showed itself. In the two days' march thirty-one died or were destroyed, twenty were abandoned from exhaustion and lameness, and seventy-two had to be sent to the veterinary hospital. *That is to say, rather more than three horses fell out every mile!*

At Springs fifty-nine remounts were received, mostly horses that had recovered and been discharged from a veterinary hospital.

From Springs we marched to Middelburg, arriving there on the 3rd August 1900.

Altogether in the march from Pretoria *via* Spring to Middelburg, a distance of 120 miles, 130 horses were sent to the veterinary hospital, mostly Hungarians, and sixty-four died or were destroyed or abandoned from exhaustion.

At Middelburg the regiment held a line of country about 12 miles from the town, remaining there two weeks, which gave the horses a rest that they badly needed. There was a 30-acre field of young green wheat close to our camp, in which we turned the horses for a short time daily, and we also went on foraging expeditions to the neighbouring farms, collecting mealies, which we gave boiled to the horses. By the time the two weeks were up the animals began to look a little less like dried up bags of bones. The only thing against them getting fit was the terrible cold at night; in fact, most of the nights were colder than any I have experienced in England. The horses had no rugs, and many had their summer coats, as they had only been landed a short time, coming from countries in which it was summer to South Africa in mid-winter. The horses that did best were those that we were able to turn loose in a high-walled kraal, as they were sheltered from the cold winds, and were able to move about and so keep warm during the nights.

We left Middelburg on the 18th August 1900, marched to Geluk, a distance of 20 miles, and received fifty-nine remounts there, mostly Hungarian horses, in a very poor condition. We remained at Geluk a few days, and had one horse killed and two wounded in a reconnaissance.

From Geluk the regiment moved to Belfast, taking part in that battle, but without any casualties amongst the horses. The night after the battle of Belfast was one of the coldest nights I ever remember; the place we camped on was about 8000 feet above the sea, and we were without blankets, cloaks, or food, with a bitter cold wind blowing. We did what the horses seemed to do every night, namely keep on the move, as the only means of inducing circulation. Next morning we moved in the direction of Waterval Boven, and from there to Carolina, where we arrived on the 6th September 1900.

The casualties for the march from Middelburg to Carolina, a distance

of 165 miles, were nine horses abandoned, ten died or had to be destroyed for exhaustion, and twenty-eight sick at the end of the march, of which eleven were cases of sore backs.

At Carolina occurred the first case of poisoning from the tulip or tulip plant. It is a plant which comes up with the first shoots of young grass on low boggy ground at the very beginning of spring, when all green food is very scarce, and for this reason is eagerly sought after by horses. It is very hard to detect when just appearing through the ground with the young shoots of grass, and this is the time that horses eat it and are poisoned. When there is plenty of green grass about, and the tulip is older, horses do not touch it. When the plant is full grown it has leaves like the common daffodil, and has a yellow flower like a tulip, hence its name. All horses eat it when young, but cases of poisoning in Colonial horses are rare, although I have met with a few. The symptoms are those of flatulent colic, with a peculiar dry, sour, musty smell about the mouth. Cases are often fatal in the course of two or three hours. The best treatment, I have found, is a hypodermic injection of half a grain physostigmin. The popular Colonial treatment is half an ounce of calcium chloride.

From Carolina we marched to Barberton, a distance of 75 miles, arriving there on the 13th September 1900, after one day's very hard fighting. The casualties for the march were two dead of tulip poisoning, one destroyed for bullet wound, and three for exhaustion; twenty-four remained sick at the end of the march, of which eleven were from sore backs. The roads were very mountainous, and we were forced to leave our transport to follow on. Luckily Barberton was full of supplies, as we had nothing with us, and it was some days before our transport came up. As it was, the horses went very short and lived on mealies.

On the 15th September the regiment marched to the Sheba mine, which is on the top of the mountain overlooking Barberton. The road to it is very steep, and we found it quite impossible to get waggons up, and only with great difficulty managed to get a Pom-Pom there. All our rations and forage were carried up by donkeys, each one carrying 50 lbs. The grazing on the top of the mountains was fairly good considering the time of the year, but the issue of forage was never more than 7 lbs. of mealies per head and often less, until 24th September, after which date forage was plentiful and a better place was found for watering the horses.

The regiment rested at Sheba until the 2nd October, on which date it began the march back to Pretoria, which was 310 miles away. On the day we left Barberton the rain began, and it rained more or less the whole way to Pretoria.

The first day we camped at the Devil's Kantoor, at the foot of the mountains surrounding the valley in which Barberton is situated. After a very cold night we started next morning to get the transport of the force up two miles of mountain road, which was axle-deep in mud. I was not in Veterinary charge of the transport, so do not know how many animals died on this piece of road, but there were mules and oxen dying and being shot (as they were too exhausted to rise when they fell) at every few yards. For two days and nights the work of getting the waggons up the mountains continued; every waggon had double or triple spans of oxen or mules, and even these

could only get a few hundred yards at a time. The road instead of improving got worse and worse, waggons sank up to the hub of the wheels and had to be dug out, blocking the road behind them. The patient oxen strained under the terrible punishment the driver is capable of inflicting with a whip, which when cracked goes off like the report of a rifle; many fell never to rise again, and were pulled out of the way, and a fresh start was made.

The pouring rain, and the howling and screaming of the drivers encouraging their teams, made the scene a veritable pandemonium. When at last all the waggons were up on the mountains, what with wet, cold, and hard work, it would have been difficult to find a more sorry-looking or exhausted lot of animals in South Africa.

By easy stages we reached Machadodorp, where we arrived on the 8th October, and rested until the 12th October. Here we received 93 remounts, mostly Colonial ponies, which did good service.

The regiment left Machadodorp on the 13th October, and marched to Carolina, arriving there on the 14th October; here again cases of tulip grass poisoning occurred, in spite of the care we took to keep horses from grazing in low ground. Twenty were poisoned, but fourteen recovered after a hypodermic injection of half a grain of physostigmin.

On the 16th October we left Carolina and moved in the direction of Lake Crissie, encountering the Ermelo Commando of Boers towards evening, and getting rather the worst of the fight; in fact, we lost seventeen horses killed and twelve missing in the action.

From this date until we arrived at Heidelberg (ten days) the Boers never for a moment ceased to worry us. It reminded one very much of a pack of small curs snapping and snarling around a big dog, but separating and running away every time the big dog turned. First of all we were taken over by the Ermelo Commando, who, as soon as we got out of their District, handed us on to the Bethel Commando, and this force in its turn passed us on to the Heidelberg Commando, who saw us safely into Heidelberg. A rear guard action was fought the whole way, a distance of 125 miles. Even at night there was no rest from the untiring Mauser. It rained incessantly, the nights were cold, and the roads were slippery and axle-deep with mud. The convoy, consisting of ox and mule transport, was several miles in length, and could scarcely get along. There was no time for grazing the oxen and no food carried for them, and this, combined with the wet and cold, killed them by hundreds, so that scores of waggons had to be burned to prevent them falling into the hands of the enemy.

We arrived at Heidelberg on the 26th October, rested there two days, and then moved on by easy marches to Pretoria, where we arrived on the 2nd of November.

The regiment I was with lost 63 horses from exhaustion alone on the march from Barberton to Pretoria, and if this is taken as average loss per regiment it would bring the total amongst the six Cavalry regiments with the force to 378 from exhaustion alone. The loss amongst the transport animals must have been very much greater than this.

Our total casualties for the march were, forty-four died or destroyed, forty-nine abandoned from exhaustion, twenty-four missing, and ninety-seven sent to the various veterinary hospitals on the road.

Of these, thirty-nine were suffering from sorebacks, forty-eight from exhaustion, and ten from other causes. We had issued to us on the march from Barberton to Pretoria 216 remounts, marching into the latter place with 224 fit horses and 48 sick.

At Pretoria the regiment went into camp and slept in tents for the first time since leaving Cape Colony, viz., ten months. The weather still continued wet.

Here we were thoroughly re-equipped and remounted up to 416 strong. The remounts were mostly North American horses, which were a decided improvement on the Hungarian horses, and did good service in spite of the fact that they were more fitted for draught than cavalry work.

On the 11th November we left Pretoria and marched by easy stages to Meyerton, arriving there on the 21st, a distance of 50 miles. We remained in this district until the 14th December, when we received sudden orders at 11 o'clock at night to march at once. By 3 A.M. we were on the march, camping at 6 P.M. near Krugersdorp, after a march of over 40 miles, having lost three horses on the road, two from fractured pastern, and one from exhaustion.

At Krugersdorp the regiment rested for four days, receiving thirty remounts.

From the 19th December 1900 until the 17th January 1901 we operated along the Magaleisburg Mountains, never doing very long marches, but moving every day. On the latter date we arrived at Johannesburg.

The total losses for this period were twenty-seven died or destroyed, of which thirteen were cases of horse-sickness. Forty-six were sent to the veterinary hospital during the march, of which twenty-eight were sore backs, and fourteen were cases of exhaustion. Forty-eight remained sick at the end of the march.

At Johannesburg we were again re-equipped and remounted up to 406 strong, mostly on North American horses. Our camp was not in a very good situation, as there was no grazing. We lost several horses from horse-sickness, infected probably on the low ground we camped on a few days before reaching Johannesburg.

On the 27th January we left Johannesburg to take part in General French's Eastern Drive, which consisted of a long line of columns, in touch with each other, and reaching from the Delagoa railway line to the Natal line, a distance of 100 miles. The columns moved each day, sweeping the country of Boers and stock.

It was now for the first time that any very serious thought was given to the welfare of the horses. They were no longer expected to carry impossible weights, wallets and all unnecessarily heavy equipment were done away with, great care was taken regarding the watering and feeding, and the animals were off-saddled at every halt when practicable. It became a crime for a man to give a horse a sore back, and the offenders marched as infantry until their horses were well again. Everything was done to make men take care of their horses, and become proud of them, and in consequence sore backs became nearly a thing of the past. Directly a horse was sick or had a sore back he was sent to the sick lines and treated, being driven along each day in a drove by Kaffirs. The animal was not sent back to duty until quite fit, and in this way a great many lives were saved, and the horses remained to do useful work for the Country. The

weather still continued wet, but the marches were not on the whole long ones.

On the 7th February we encountered the Boers, and had three horses wounded during a charge, in which the regiment for the first time got at the Boers with the sword, taking their camp and a number of prisoners and ponies, the latter of which did good service for us afterwards.

We now moved on towards Piet Retief, arriving there on the 16th February. Here we expected a convoy with supplies to meet us, but no sooner did we get to Piet Retief than all the rivers became in flood through the heavy rains, and we could neither get our convoys of provisions to us, nor go to them.

From the 16th February until the 11th March, a matter of four weeks, we remained between Piet Retief and Paul Peitersburg, building bridges and pontoons and mending roads, in one continuous downpour of rain, and only just getting enough food through to keep the men going. The only thing there was enough of was beef, as we had a lot of captured oxen with us. The horses lived on a few pounds of mealies when we were able to get them, and green mealie stalks. The road from Piet Retief was one long line of dead oxen and mules that had died from exposure to wet, cold, and hard work, in trying to get the convoys to us over a nearly impassable road.

But, like everything else, the rain in the Pongola valley must have an end, and on the 24th March we marched towards Vryheid, and then on to Glencoe in Natal, where we entrained our horses on the 3rd April 1901 for Belfast in the Transvaal.

Entraining horses in South Africa consists in packing loose horses as tightly as possible in a closed or open truck, and it is not at all a lengthy process; in fact, a regiment can entrain in two hours with ease. The horses are fed and watered in nose bags during the journey, the men in charge of them climbing up the outside of the truck, and reaching the horse's head through the bars. If a horse slipped down he had to remain down until the end of the journey, unless the train happened to pull up alongside a high platform, when the other horses were got out and the fallen ones released. Injuries and even death from being trampled on often occurred.

We left Glencoe by train on the 4th April, and arrived at Belfast on the 7th, a distance by rail of a little over 300 miles. For the three nights and four days the majority of the horses remained in their trucks, only a few being got out on the second night. On arrival at Belfast the horses looked as if they had not had food for days, and were so stiff that they could hardly walk; it took a long time for them to recover from the effects of the rough journey.

Before entraining at Glencoe there was not a single case of mange amongst the horses, but on arrival at Belfast one truck load was badly affected, and had bitten and rubbed themselves nearly raw. From this date the horses of the regiment were in a more or less mangy state for some months; the disease spread rapidly in spite of all the care we took to isolate the affected ones, and everything seemed against our stamping it out.

As it was very cold the horses had to be rugged up at night, and it was very hard when camping after dark to make sure that every horse had his own blanket, or to prevent them being rolled up together when packed in the early morning. Later in the year, when we were able

to do without horse rugs, we practically stamped mange out ; but new horses joining the regiment, even when great care was taken to see they had no mange about them, would develop it, and start the disease afresh.

The treatment adopted was to regard every horse in the regiment as an infected animal, and have them all washed with izal solution twice a week, while the actually mangy animals were dressed with kerosine oil 2 parts, linseed oil 1 part, soap solution 2 parts ; or, creasote 1 part, linseed oil 20 parts, soap solution 30 parts. This mixture is applied all over the animal, and he is groomed every day and never washed ; every third or fourth day he is redressed, and in two or three dressings has quite recovered. The animal can be worked the whole time if necessary, as with grooming he gradually works quite clean again. Washing with any antiseptic in bad cases of mange only hardens the skin, and it does not penetrate like oily dressings. Linseed oil if applied without being mixed with soap solution turns into varnish in the sun, and it is quite impossible to remove it until the animal changes its coat.

At Belfast the regiment rested to get the horses fit again after the railway journey, and received 100 remounts, mostly North Americans in very bad condition. The nights now were very cold, and horse rugs were issued, which, although they increase the size of the convoy, being an extra 12 lbs. per horse to be carried on the waggons during the day, add very materially to the efficiency of the force in keeping the horses in better condition.

On 16th April we left Belfast, and marched by easy stages to Roos Senekal, a village north of the Delagoa railway, and just on the border of the Boer Veldt. We arrived there on the 23rd April, remaining around that district until the 29th, when we left, marching west in the direction of Middelburg, but only to return the following night, after making a night march, surrounding Roos Senekal at daybreak, and taking a good many Boers who thought we were safely on our way to Middelburg.

From Roos Senekal we marched by easy marches to Middelburg, arriving there on the 6th May.

The regiment left Middelburg, Transvaal, on the 12th May with 420 horses, of which twenty-five were Boer ponies received on the day of marching. We moved in the direction of Steynsdorp on the Swaziland border, a distance of 167 miles, reaching our destination on the 30th May. On the march we lost twenty-one horses, ten of which were from horse sickness, and three destroyed for bullet wounds received in a skirmish with the enemy. We rested at Steynsdorp one day, and then started on our return march to Middelburg, arriving there on the 3rd July.

Our loss of horses on the return march was forty-six, thirteen of which were lost from exhaustion in one day, in a very long reconnaissance of 40 miles.

The whole march to Steynsdorp and back was very uneventful ; we had infantry with us, and did very short marches when not on reconnaissances. The distance from Middelburg to Steynsdorp and back was 335 miles, and the total losses were sixty-seven died or destroyed, and twenty-two sent to hospital.

From Middelburg the regiment moved by easy stages along the line to Pretoria, a distance of 85 miles, arriving there on the 14th July.

We again left Pretoria on the 22nd July, entraining for Vredefort Road Station in the Orange River Colony, and marched from there to Heilbron, where we joined Col. Rimington's column. From this date until the end of December we operated around Heilbron. The work consisted in making sudden dashes from the Heilbron district, surprising Boer laagers, then back to the line again for supplies, followed by one or two days rest, and out again once more. The majority of the marching was done at night. The column in this way would cover 30 miles before daybreak, then make a rush after Boers, and back to the line again. I have known the column to do five night marches running, sleeping during the day, at least those who could sleep in a burning sun, often without any shade, did so. The horses were kept in good condition, having every care bestowed upon them. When not after the Boers, the men walked and led their horses nearly as much as they rode them. The great majority of the horses in the regiment at this time were Colonials. We had some Hungarians issued to us, but we exchanged them, as we found them quite useless, and received Colonial horses in their place.

We only left the district once during this period of six months, and that was on the 8th November, when the column, with only the pick of their horses and a very light convoy, moved to the Ermelo district to try to catch General Botha. We made a night march from a camp near Ermelo, catching a Boer dispatch rider, from whom the whereabouts of Botha was learnt. We marched there, and arrived at daybreak in a thick mist which suddenly lifted, but Botha had received warning of our approach and just got away. It was here the regiment lost the last horse remaining out of those they left England with; it was ridden by a trumpeter the whole time, and had never been sick. It was shot dead by a chance shot from the Boers.

We now returned to the Heilbron district again.

Our total losses for this portion of the war, 22nd July to 31st December 1901, were 313 died or destroyed, 38 missing, and 314 sent to the veterinary hospital. The distance travelled was 1814 miles. The losses were chiefly due to the very long distances travelled, a day and night march often amounting to 50 miles, and some of this at a fast pace. When a horse became done up the man had to be put on a spare horse, and if his own was unable to keep up it had to be shot. As the column was a mobile one, the Boers never quite knew where to expect it, and were never safe within 40 miles of it.

From January 1902 to the end of the war, 31st May 1902, the work was chiefly drives. Columns formed up reaching from one Block House line to another, always in touch with each other, and their picquets only a few yards apart at night, in one continuous line. Every day the line advanced, rarely doing very long marches, until the Boers and stock, if they did not break through, were driven against a Block House line and captured.

The horses had by far the easiest time they ever had during the war. The total losses for this period were 164 died or destroyed, and 204 sent to hospital. The distance travelled was 1610 miles.

When Peace was declared the regiment was at Heilbron, and marched to Bloemfontein, a distance of 160 miles, without any casualties, arriving there on the 29th June 1902, with 540 fit horses.

This brings to a close the rough notes of a memorable campaign, the total losses of which are best summarised as follows :—

From November 1899 to June 1902 the regiment I served with used up 3750 horses. The distance travelled in a straight line was 6116 miles; but, as this does not include reconnaissances and scouting, these would in all probability double the mileage, bringing it up to 12,232 miles. On this basis, which is a liberal one, we used up one horse every $3\frac{1}{2}$ miles during the campaign.

I hope next time we may do better.

TABLE No. 1.—*Showing Strength of Regiment on Landing, Number of Horses that Joined, and the Number Expended, from November 1899 to 30th June 1902.*

Strength of regiment at landing	406	
Number of remounts that joined	3061	
Horses picked up on the veldt	222	
Transfers from other regiments, etc. . . .	601	
Total	4290	Horses joined . 4290
Horses sent to veterinary hospitals	1600	
Horses missing	201	
Horses returned to remount dépôt	202	
Horses abandoned	201	
Horses destroyed	595	
Horses that died	482	
Horses transferred to other units	469	
Total	3750	Horses expended 3750
		540 Remaining.

TABLE No. 2.—*Showing the Sick and Lane Horses, from November 1899 to 30th June 1902.*

<i>Diseases, etc.</i>	<i>Admitted Sick.</i>	<i>Total.</i>	<i>Cured.</i>	<i>Sent to Vet. Hospitals.</i>	<i>Abandoned on March.</i>	<i>Died.</i>	<i>Destroyed.</i>	<i>Remaining Sick.</i>	<i>Total.</i>
General diseases	1993	1993	249	877	201	203	463	—	1993
Respiratory apparatus	57	57	5	3	—	46	3	—	57
Circulatory	4	4	1	—	—	1	2	—	4
Urinary	1	1	—	—	—	—	1	—	1
Generative	3	3	1	2	—	—	—	—	3
Digestive	98	98	47	1	—	38	12	—	98
Liver and spleen	4	4	3	—	—	1	—	—	4
Nervous apparatus	16	16	—	3	—	3	10	—	16
Tegumentary ¹	23	23	11	12	—	—	—	—	23
Locomotor apparatus	396	396	132	196	—	1	60	7	396
Zymotic diseases ²	12	12	5	1	—	4	1	1	12
Visual apparatus	11	11	3	8	—	—	—	—	11
Surgical diseases and accidents	1309	1309	626	656	—	2	21	4	1309
Wounds, shell	3	3	1	—	—	2	—	—	3
Wounds, bullet	163	163	16	21	—	119	7	—	163
Killed by lightning	4	4	—	—	—	4	—	—	4
Drowned	1	1	—	—	—	1	—	—	1
Horse sickness	72	72	—	—	—	57	15	—	72
Total	4170	4170	1100	1780	201	482	590	12	4170

¹ Only horses that were too bad to work are shown as having mange.

² Amongst these is included only one case of glanders; this does not, however, represent the total number of cases in the campaign. There are twenty-five horses in the class "General Diseases," which were destroyed at various times with "Clinical Symptoms" while suffering from other affections. I was struck by the infrequency with which glanders occurred.

TABLE NO. 3—Showing Casualties for each March, and Distance Travelled.¹

Time Occupied.		Strength at Date of Marching.	Died or Destroyed on March.	Abandoned on March.	Missing.	Sick at End of March.	Fit at End of March.	Distance Travelled in Miles.	Station.		Remarks showing Principal Causes of Casualties.
Date From.	Date To.								From.	To.	
10/12/99	6/3/00	490	60	NIL	13	41	376	80	Arundel	Coleburg	31 killed in action; 30 destroyed, bullet wounds; 15 sore backs.
6/3/00	17/4/00	398	4	NIL	NIL	93	373	300	Coleburg	Bloemfontein	3 destroyed from exhaustion; 1 died; 10 sore backs remaining.
11/2/00	14/2/00	94	5	NIL	NIL	10	79	80	Mokkera	Kimberley	5 destroyed, bullet wounds; 6 sore backs remaining.
11/2/00	13/3/00	79	4	9	NIL	20	46	100	Kimberley	Bloemfontein	Exhaustion cause of casualties; 12 sore backs remaining.
6/3/00	12/3/00	417	48	2	NIL	63	323	120	Bloemfontein	Kroonstad	31 killed in action; 14 died of wounds and exhaustion; 11 sent to veterinary hospital; 23 sore backs remaining.
20/5/00	6/6/00	372	30	70	1	72	196	175	Kroonstad	Pretoria	10 died; 20 destroyed from exhaustion; 70 abandoned; 30 sore backs remaining.
9/7/00	3/8/00	556	41	23	37	24	323	120	Pretoria	Middelburg	108 sent to veterinary hospital on march, mostly sore backs and exhaustion; 18 died and 23 destroyed, exhaustion.
18/8/00	6/9/00	321	10	9	5	28	306	165	Middelburg	Carolina	59 remounts joined; 22 horses sent to veterinary depot, of these, 11 were sore backs, 3 exhaustion; 19 died and destroyed, exhaustion; 11 sore backs remaining.
10/9/00	14/9/00	380	6	8	6	24	286	70	Carolina	Barberton	Tulip poisoning, 2; bullet wound, 1; destroyed, exhaustion, 3; sore backs remaining, 11; exhaustion, 8.
8/10/00	3/11/00	271	44	49	24	48	224	310	Barberton	Pretoria	216 remounts joined; 97 horses sent to veterinary hospital on march, of which 39 were sore backs and 48 exhaustion; 92 died or abandoned, exhaustion; 24 sore backs remaining.
11/11/00	21/11/00	416	6	NIL	9	10	391	55	Pretoria	Meyerton	Sore backs, 2.
14/12/00	16/12/00	377	3	NIL	NIL	1	373	40	Meyerton	Krugersdorp	1 died, exhaustion; 2 destroyed, fracture.
19/12/00	16/1/01	406	37	NIL	5	48	343	297	Krugersdorp	Johannesburg	20 remounts joined; 46 horses sent to veterinary hospital on march, of which 25 were sore backs and 14 exhaustion; 18 cases of horse-sickness; 18 sore backs; 16 exhaustion cases remaining end of march.
27/1/01	6/5/01	404	112	NIL	25	20	405	350	Johannesburg	Middelburg	156 sent to veterinary hospital, consisting of 14 sore backs, the remainder exhaustion and debility; of those, 25 died horse-sickness, 6 destroyed; the remainder died and destroyed exhaustion.
12/5/01	30/5/01	430	21	NIL	10	20	454	160	Middelburg	Steynsdorp	71 remounts joined, and 13 Boer ponies picked up on march; 20 horses sent to sick depot.
1/6/01	8/7/01	454	46	NIL	NIL	23	394	175	Steynsdorp	Middelburg	8 Boer ponies taken on strength; 22 sent to sick depot.
8/7/01	14/7/01	395	2	NIL	NIL	8	385	85	Middelburg	Pretoria	767 remounts joined; 91 ponies taken on strength; 40 resolved from A.P.M. column; 314 sent to sick depot and 99 to remount depot.
22/7/01	31/7/01	385	313	NIL	38	314	519	1314	Orange River Colony	Orange River Colony	338 remounts joined; 308 horses sent to veterinary hospital; of those died and destroyed, majority were exhaustion.
1/1/02	18/5/02	519	164	NIL	9	208	496	1610	Orange River Colony	Orange River Colony	
21/6/02	29/6/02	540	NIL		NIL	NIL	540	160	Heilbron	Bloemfontein	

¹ This table only shows the actual casualties during the marches, and does not include those occurring in the intervals between the marches.

PYROPLASMOSIS OF THE DONKEY.¹

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THE diseases of animals due to the presence of protozoa in the blood have recently assumed so much importance that a special class—pyroplasmosis—has been adopted for them by the French veterinarians. Nearly all the domesticated animals are now known to suffer from one or more diseases of this class. Up to the present, however, I am unaware that any writer has described a pyroplasmosis in the donkey.

My first acquaintance with the disease dates from the 2nd of March of this year, when I was sent to investigate an outbreak of disease among donkeys at Lydenburg, which was attended with heavy mortality. I made eight *post-mortem* examinations on the day of my arrival, and the appearances were so marked, so much alike in every case, and so different from anything I had seen before, that I came to the conclusion they were suffering from an unknown specific blood disease. Since then I have had outbreaks all along the Waterburg district, among isolated spans belonging to settlers and farmers, and also among a large mob up the Aapies River.

The animals attacked do not appear to belong to any particular country, breed, age, or sex, but Colonial-bred donkeys in most cases pass easily through the first or acute stage, and then recover without passing on to the second or chronic form; in fact, in some cases, were it not for the extremely high temperature, coupled with the fact of other animals in the same mob or span being affected, no suspicion of disease would arise, although a careful observer might notice the animal a little dull and off-colour.

From this it will appear that native donkeys are more or less immune; for, although I have never seen one escape the disease if placed under the same conditions as imported donkeys, I have also never seen one die from it.

The disease does not appear to be congenitally transmissible. Foals of dams suffering from the disease in either of its stages appear to be quite healthy. Although the mother may be a mere bag of bones, the offspring thrives and does well, microscopic examination of blood smears revealing no pyroplasms, and intravenous injection of blood from an infected animal causing no reaction. This latter circumstance, however, must not be taken as conclusive evidence of immunity, as the intravenous injection of virulent blood into healthy adult donkeys, mules, a dog, and rabbits, caused no systemic disturbance whatever, thus by analogy leading us to the conclusion that there must be an intermediate host or carrier.

Symptoms.—The period of incubation is not known. The disease is usually ushered in by symptoms of a very trivial character; in fact, in the acute or first stage the disease is very difficult to diagnose, especially at the outset, when one has not had previous cases and *post-mortems* to give a clue.

The animal usually stands apart from the others, and seldom lies down during this stage of the disease. It appears dull, sleepy, and stumbles when walking; the skin is hot, and there is thirst and often

¹ A paper read before the Transvaal Veterinary Medical Association.

slight loss of appetite. The visible mucous membranes are clean, pale, and blanched, but not yellow or dirty as in equine malaria. This also applies to the conjunctival membranes, but the membrana nictitans is often the seat of dark red patches of ecchymosis. A general or localised erythema usually makes its appearance during the first four or five days, with a vesicular eruption along each side of the spine, and occasionally on the muzzle, nose, and legs, below the knees and hocks; the vesicles afterwards become pustular, dry up, and desquamate. The pulse is usually full, quick, and intermittent, but varies a great deal. The temperature is always very high in the acute stage, ranging from 105° to 107° F., generally over 106°, and lasting usually five or six days, when it drops suddenly to sub-normal, where it remains until death or recovery. At the same time there is good reason to suppose that in some cases the period of fever is of longer duration; in one case, at least, the animal had a temperature until the ninth day, when it dropped suddenly, the patient dying a few hours later. In spite of this high temperature, it is remarkable that there are no rigors, that the extremities, legs, ears, etc., are always warm, and that the respirations are not increased; there is no swelling or œdema of the sheath or extremities, no pain or colic, and no loss of flesh, although a marked change comes over the patient as soon as the temperature drops.

In the second, or chronic form, the disease is characterised by progressive anæmia and rapid loss of flesh, especially in the muscles of the back and hind-quarters. The animal walks in a straight line, as though trying to balance himself on a tight-rope, and looks neither to the right nor to the left; he often appears to be in a state of semi-coma. In some cases a partial paralysis of the hind-quarters and sphincter ani, with consequent dilatation of the anus, is present, due, no doubt, to interference with the normal functions of the cord in the lower dorsal and lumbar regions, as the result of pressure caused by the exudate which is present in greater or lesser quantities within the spinal membranes.

The animal now becomes a sorry spectacle, for, in addition to the extreme emaciation, the hair falls out, and the skin peels off wherever there was rash. The action of the heart is irritable, irregular, and often tumultuous, the palpitations being heard some distance from the animal, and, in fact, sometimes so violent as to cause the whole frame of the animal to shake. The pulse may be full and quick, weak and thready, or intermittent, but is of no diagnostic value whatever, as it entirely depends on the extent of the heart lesions. The breathing is quickened, but each individual respiration is shallow, and the acceleration is probably due to heart disturbance, as it is very unusual to find the lungs in anything but a normal condition.

The appetite will, during this period, have increased if anything; in fact, in many cases the animal has a voracious appetite and will eat to the last. The evacuations are little changed in character. There is a copious excretion of urine, which is usually very pale and watery; but in a small percentage of cases the condition of hæmoglobinuria is present.

Unless treatment is prompt and energetic, the animal gradually wastes away; the anæmia becomes more marked, the animal appears half asleep, has to be helped on to its legs in the morning, ultimately

getting so weak that it lies prone for probably a couple of days, and dies without a struggle. In other cases complications may arise; and bowel troubles with colic occur occasionally, due to the animal being unable to digest the result of an appetite which he is always ready to satisfy.

The different kinds of pneumonia, but principally those of a septic infection, also hasten the end, and I have also found inflammation of the bowels to be the immediate cause of death; but these have nothing whatever to do with the disease, and are due to the conditions under which the animals are placed when in such a low and debilitated state.

From this it will be seen that no symptom or group of symptoms is really specific, and diagnosis in the first or acute stage is extremely difficult. At the same time, the fact of the symptoms being of a negative character, coupled with the high temperature, gives one a clue. Blood smears may confirm, or they may not, as the pyroplasma is not always present in the acute, and never in the second or chronic stage.

Post-mortem Appearances.—In the first or acute stage there is usually nothing to mark externally; internally, one cannot help noting the sort of parboiled appearance of the majority of the tissues. The liver is usually very much congested, with a yellow tinge, and its substance is also more or less broken down, but there is no jaundice or staining of the tissues.

The spleen is enlarged, but there is no breaking down of its structure; the kidneys are pale; all the other organs are more or less normal, and one is at a loss to explain death except by the action of a toxic poison.

The *post-mortem* appearances of the second or chronic stage are, however, more marked. Externally, extreme emaciation is noted, pallid visible mucous membranes, and a more or less bare condition of the skin along each side of the spine, and down each hind-quarter. On opening the animal it will be noted that there is marked anæmia, extreme pallidity of all the tissues, with sometimes an enormous serous effusion into the abdominal cavity—in fact, at times there appears to be an acute dropsy. This effusion is not confined to the peritoneal cavity; there *may* be a slight effusion into the pleural cavity, but there is *always* an abnormal quantity of fluid in the pericardial sac, the liquid being blood-tinged in a small percentage of cases.

In addition to petechiæ being found on the membrana nictitans, ecchymoses are occasionally found on the different mucous and serous surfaces, and D. V. S. Conacher states that he found hæmorrhagic patches in the intestines; but, as I have come across very few cases out of a very large number of *post-mortems*, and as I have already stated that I consider inflammation of the bowels to be a concurrent accidental condition, I have not included it in either *ante-* or *post-mortem* symptoms. There is no jaundice. The changes in the heart are most marked; the pericardium invariably contains an amber-coloured fluid, which in some cases is a deep port-wine colour. The heart itself is very flabby and enlarged, and the fat at its base and along the auriculo-ventricular furrow is replaced by an almost transparent gelatinous exudate, but it may be amber or pale-green in

colour. Although this condition is not specific to the disease under notice, it is invariably found, and is therefore of considerable diagnostic value.

Subendocardial ecchymoses are also often found, and it is very rarely one makes a *post-mortem* examination without finding *ante-mortem* clots in the heart and extending along the course of the large blood vessels; and this clotting is so perfect that by gentle traction one may pull out the clot for a considerable distance, showing the branching of the various vessels which they occupied. I have also found extensive *ante-mortem* clots in the blood vessels of the hind limbs. The blood itself is noticeably pale in colour, and when the hand is dipped into it it runs off without leaving any stain. If placed upon a slide or a piece of writing paper, the red corpuscles immediately run together in nests or groups, so that they become visible with translucent liquor sanguinis between.

The larynx, trachea, bronchi, pleura, and lungs present a perfectly normal appearance, but occasionally symptoms of bronchitis are present, and I have found several cases of septic pneumonia, either running concurrently with the disease under notice, or induced by the lowered state of the general health and the more or less insanitary conditions.

Nodules are also found occasionally in the lungs, due no doubt to some nematode which has undergone calcareous degeneration, but this has nothing to do with the disease.

The lymphatic glands throughout the body are more or less swollen, and often show a hæmorrhagic infiltration. The mouth, pharynx, and œsophagus present no abnormal appearance.

Although there is usually an enormous serous effusion into the peritoneal cavity, there is no inflammation, either circumscribed or general; it is purely a transudate.

Out of the enormous number of *post-mortems* that I have made, I have never found any abnormality of the stomach, but in one case examined by D. V. S. Conacher he found inflammation of this organ, which may have been an accidental occurrence.

The intestines are as a rule more or less normal, but pale in colour; they often contain enormous quantities of nematode worms (*Sclerostoma equinum* and *Sclerostoma tetracanthum*), so numerous, in fact, that one might easily jump to the conclusion that the presence of the worms was the cause of the disease, but it is not so. All parasites, whether internal or external, thrive and multiply when the body juices of their host lose their protective action from disease or debility, and the worms in question simply revel in the catarrhal condition of the mucosa which is nearly always present.

In the earlier stages of the disease the liver is always enlarged, and often presents yellowish-white patches on its surface, which appear to be due to thickening of the capsule; but, as the disease assumes a chronic form, cirrhosis takes place, with consequent contraction in at least 75 per cent. of cases.

In several cases I have found liver flukes in great numbers, and in a few cases the liver was simply studded with calcareous degenerations of cestode larvæ.

The spleen is always enlarged and pulpy, often broken down, and

may weigh anything up to 7 lbs. ; in fact, its naked eye appearance is that of an anthrax spleen.

The kidneys are always pale, and present a parboiled appearance.

The bladder on *post-mortem* is always enormously distended, usually with pale watery urine, but occasionally with blood-coloured contents, the organ itself presenting no abnormality.

The brain, as a rule, presents a perfectly normal appearance, except that there is usually fluid in the lateral ventricles, which is colourless or slightly tinged. There is nearly always an increase of fluid within the spinal membranes, and occasionally a gelatinous deposit in the lower dorsal and lumbar regions. These conditions probably account for the semi-coma and partial paralysis so often seen in this disease.

Cause of the Disease.—The cause of the disease was discovered by Dr Theiler in blood smears made from donkeys during the first outbreak, and I quote his report in his own words :—

“The cause of the disease is the presence of an endoglobular parasite invading the red blood corpuscles. It belongs to the class of pyroplasma, and is very closely related to, if not identical with, the pyroplasma found in the malarial or biliary fever of the horse and mule.

“The pyrosoma, as it was originally called in cattle, belongs to the same group, as also does the pyroplasma of biliary fever in the dog. The pyroplasma of the donkey is round in shape, and varies somewhat in size, from about one sixth to one third of the red corpuscle in which it is found ; pear-shaped or oval forms are exceptional.

“In staining with ordinary aniline dyes—methylene-blue, for instance,—the pyroplasma is recognised as a blue disc, on the green-tinted corpuscle.

“In staining with eosin-methylene-blue mixture, specially known as Laveran's stain, and in mixtures of Azur II. with eosin, the finer structure of the pyroplasma is recognised. When stained, a nucleus is seen which takes a red tinge. Around the nucleus (karyosoma) the faintly-bluish protoplasm of the parasite cell is recognised. Sometimes a clear zone encircles the nucleus. The karyosoma may be recognised in all forms of the pyroplasma, and varies according to the size of the latter.

“The largest forms which we recognise are apparently those which multiply by fission.

“Thus we recognise in one protoplasm four karyosomas, and in a later stage we find each four karyosomas separated, and surrounded by a small zone of bluish-tinged protoplasm.

“This form resembles a rosette and is very typical. In still later stages we find these rosettes completely separated ; the newly formed pyroplasms leave the red corpuscle, and thus it happens that we find two parasites in one cell. We may, however, find three individual pyroplasms, which are either completely separated from each other, or still hanging together. I have not been able to trace the pyroplasma outside the red corpuscle, but there is no doubt that it exists in the plasma, as it does in biliary fever of the horse.

“The pyroplasma is usually found in acute cases. It may be in the blood, the heart muscle, or the liver, but it is found most abundantly in the spleen, where the forms of multiplication—the rosettes—are most frequently met with. So much does the pyroplasma resemble

in its form and in its reproduction the pyroplasma found in horses and mules suffering from biliary fever, that one is struck with the idea that probably they are the same species, or varieties of the same species of endoglobular parasite."

Experiments.—Very little experimental work has been carried out, as those of us who have come in contact with the disease have had our hands too full to devote the necessary time and close observation demanded in research of this nature, and what little has been done is of a negative character.

I.—A fairly young large Kaffir hound was inoculated at Lydenburg by D. V. S. Conacher and myself, with blood from a donkey owned by the P. W. Department, suffering from the disease in the acute stage, with a temperature of 106.4° F., pyroplasms being afterwards found by Dr Theiler in blood smears obtained at the time of injection. The dog was forwarded to Dr Theiler for observation. No reaction and no systemic disturbance took place; in fact, the dog improved in condition, and no pyroplasms were found, although the blood was examined daily for an extended period. The donkey from which the blood was taken ultimately died, and D. V. S. Conacher, who made a *post-mortem*, found the usual appearances.

II.—Two rabbits inoculated with blood by Veterinary-Surgeon Pye at Lydenburg were sent to Dr Theiler for observation; no reaction took place.

III.—Three mules inoculated by myself with 50 cc. of blood from donkeys in the second or chronic stage of the disease, and suffering from its sequelæ, caused no elevation of temperature, and no systemic or local reaction.

IV.—A healthy donkey was injected by myself with 20 cc. of blood from a donkey in the second stage of the disease, the injection being made into the jugular; and no reaction whatever took place.

V.—Two healthy donkey foals, whose dams had suffered from the disease and recovered, were inoculated by me with 20 cc. of blood from a donkey in the chronic stage of the disease; no reaction whatever took place. No blood containing pyroplasms was available at this time, as all the mob were in the second stage.

VI.—Struck by the similarity of many of the *post-mortem* appearances of this disease and tick fever in cattle, allowing for difference in species, D. V. S. Conacher was induced to inoculate a healthy donkey with 50 cc. of virulent blood from a certified case of Rhodesian tick fever. The second day the temperature rose to 103° F., then fell to 100.3° F., and ranged between that and 101.4° F. until the sixth day, when he was unable to continue his observations through stress of work.

I am afraid these experiments tell us very little, except that the disease is not contagious in the ordinary way; but, by analogy, we can assume that the hæmatozoon undergoes some metamorphosis, probably in the body of a tick by which it is carried and the disease transmitted, as has been proved to be the case in red-water and the malarial biliary fever of dogs.

At one time I was of opinion that this disease was quite different from anything found in the horse or mule, but I have lately had occasion to modify my views, for not only have I seen something very similar, if not identical, in mules, and which I have not had time so

far to investigate, but it will be seen that in many things the disease resembles equine malaria. The hæmatozoon is of the same type, the disease runs a very similar course, and although the clinical symptoms, especially in the acute form, vary considerably, this may be accounted for by difference of species. At the same time, although they are so very similar, I am of opinion that they are different and distinct diseases, caused by blood parasites that are possibly as nearly related to each other as the horse is to the ass. One reason for coming to this conclusion, and a very important one I think, is that I have inspected mobs of donkeys that were either healthy or suffering from worms, or some other complaint, and been called in to attend cases of biliary fever among the conductors' horses used for herding the donkeys. These were invariably imported horses, but so were the donkeys.

At the present time I am treating a mob of 1000 donkeys suffering from pyroplasmosis, but the horses, to the number of a dozen or more, that herd them, mix with them, eat the same grass, drink the same water, and get bitten by the same tick, are not only quite healthy but are in the pink of condition. This also applies to the mules used for transport on the same depôt.

On a farm at Hartingsburg, near Warm Baths, I had twenty donkeys affected with this disease, eight being Argentines and twelve South African bred. One, an Argentine, died in the acute stage before I arrived; another was very bad in the acute stage, with a temperature over 107° F. My prognosis was that he would die, but he recovered with very little evidence of sequelæ. The other six had passed on to the second stage and had sub-normal temperatures; they all recovered and are now at work. The twelve country-bred donkeys had all either been sick, or had high temperatures when I examined them; and, as is usually the case with native donkeys, passed through the acute form, recovered, and were the first to be at work. The only horse on the farm was never ailing for an hour; but though it was country bred so were twelve of the donkeys, and, if the diseases are caused by identical pyroplasms, one would expect the horse to show it more than the donkeys, seeing that the symptoms of biliary fever of the horse are so much more marked during the acute stage.

From this it will be seen that it is possible to have horses affected with equine malaria in contact with a herd of donkeys, and the donkeys not to be affected with the pyroplasma equi. It will also be seen that it is possible to have 1000 donkeys suffering and dying with heavy mortality, and the horses which herd them apparently possessing immunity.

Treatment.—The one redeeming feature of this disease is that it is amenable to treatment; prognosis, except where the animal has suffered from the disease for some time and where organic changes have taken place, is always favourable. It is essential, however, that the animal be treated as an invalid; he must have generous fare, food that he can easily digest, mash made with boiled oats and maize, with bran added until it is crumbly moist, and green food if it is obtainable.

In the first or acute stage, until the temperature drops, I have found the administration of 2 drachms of either ammonia carbonate or chloride, combined with 2 drachms of sodæ hyposulphite, to be most

effective ; and if the heart is irritable, irregular, or tumultuous, as it often is, I have found a drachm of belladonnæ extract, given as an electuary, or combined with the ammonia and hyposulphite of soda, to have a markedly beneficial effect.

Immediately the fever abates, change the medicine to 3 grains of arsenic in a drachm of sulphur, given dry on the tongue once a day for ten days ; then do nothing for two days, after which start the administration of tonics. I have found a drachm of finely pulverised ferri sulph. placed on the tongue in the same manner as the arsenic mixture to act very well. I do not advise the administration of purgatives, even at the commencement of the attack ; but, if the animal is stall-fed, a couple of ounces of mag. sulph. in a bucket of water, left before the patient so that he can drink whenever he wishes, is a great help in keeping the bowels in order, and also acts as a febrifuge.

Other drugs have been experimented with, and with success, especially carbolic acid, quinine, and salicylate of soda ; but these drugs are not so convenient to use, especially if one has a large mob to dose. They are more expensive, and their exhibition has not proved so effective in my hands as those recommended.

In conclusion I have to thank D. V. S. Conacher for much assistance, and I am also under the greatest obligation to Dr Theiler for his generous help and advice, for which I am grateful.

REPORT ON EXPERIMENTS WITH ANTI-RINDERPEST SERUM.

By STEWART STOCKMAN, M.R.C.V.S., Principal Veterinary Surgeon to the Transvaal Government, Pretoria.¹

IN accordance with instructions, I requested Dr Lingard to inform me regarding his plan of experiments which were to be carried out in the Madras Presidency with a view to ascertaining the correct dose of anti-rinderpest serum.

On the 15th December 1902, in obedience to orders, I proceeded from Khandwa to Madras, to consult with Vet.-Major Gunn, Supt. C. V. D., about the arrangements.

Major Gunn had arranged that the Taheildars of the Taluga in which experiments were to be performed should get farmers to lend the requisite number of cattle on condition that they would be compensated for any loss. We decided, however, to purchase all animals to be used as controls and for the upkeep of virulent blood, as it was thought possible that the death rate amongst these animals might be high. This arrangement led to a very considerable saving, and made it possible to undertake test experiments on a much larger scale than was originally intended. I had been given to understand that my service in India should not cease until the experiments were finished, but unfortunately it was impossible to complete the whole series, as I was informed later that my departure for the Transvaal should not be unnecessarily delayed.

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I received from Dr Lingard a number of sterilised flasks for collecting and defibrinating blood, and the serum which he desired to be used—Serum XI. Serum XI. in the last Report of the Imperial Bacteriologist, 1901-02, is standardised at 8 cc. per 600 lbs. for plains animals. This means, according to the method adopted at Muktesar, that 144 cc. of Serum XI. is the dose required to immunise a hill animal, therefore eighteen times less—8 cc.—is sufficient for cattle of the plains. This method of standardising serum is open to grave objections, which will be apparent on consulting the results of the experiments.

The plan of experiments sent to me by Dr Lingard were briefly as follows :—

I. *Pure Breeds.*—To utilise specimens of each typical breed of cattle found in the Province.

II. *Mixed Breeds.*—To obtain a number of animals either from the bazaars held in the district, or, when available, from Government farms and dairies, irrespective of their breed.

III.—To obtain twenty-one cattle of each breed and initiate experiments by the simultaneous method, as follows :—

				Dose of Serum 600 per lbs.	Virulent Blood Test.
3	cattle to receive	.	.	5 cc.	1 cc.
3	"	"	.	6 cc.	1 cc.
3	"	"	.	7 cc.	1 cc.
3	"	"	.	8 cc.	1 cc.
3	"	"	.	9 cc.	1 cc.
2	"	"	.	10 cc.	1 cc.
2	"	"	.	15 cc.	1 cc.
2	"	"	.	None, control	1 cc.

IV.—To calculate the weight according to the formula $G.2$ (girth of chest in inches behind shoulder) \times L. (length in inches from point of shoulder to buttock under hip joint) 300—weight in lbs.

Where possible, to obtain the actual weight of animals by a weighing machine.

It was not possible to get access to a weighing machine in any of the districts visited, but this is only what one must expect when operating in the field.

The plan of these experiments is identical with that followed at Cawnpore, Poona, and Nagpur in 1902 (Report of Imperial Bacteriologist 1901-02). The animals experimented with at the above places received varying doses of serum, followed immediately by 1 cc. of virulent blood in each case. It was concluded at Cawnpore that 10 cc. of Serum XI. (per 600 lbs.) by serum alone method would in an ordinary outbreak be sufficient to ward off the effects of the disease.

At Poona it appeared from the results of the experiments that 8 cc. (per 600 lbs.) would prove ample to protect the Sindh breed by the simultaneous method. In the event of an outbreak it is stated that 15 to 20 cc. by serum alone method should prove sufficient.

Six buffaloes (five Gujrat and one Jafferabad breed) received doses of from 5 to 7 cc. of serum (per 600 lbs.). They took the disease when inoculated with virulent blood, but recovered, and it is con-

cluded that 20 cc. of same serum by serum alone method should protect for three or four months.

At Nagpur 7 cc. of Serum XI. (per 600 lbs.) was sufficient to protect in the case of the simultaneous method, but several animals showed symptoms of rinderpest after inoculation with varying doses of serum, while five control animals which had received no serum did not succumb although inoculated with doses of virulent blood varying from 3 to 10 cc. It is concluded from this that 15 to 20 cc. (per 600 lbs.) of serum by the serum alone method will protect for a period of several months.

In these experiments the animals, as is the case in all countries where rinderpest has become permanently established, presented a high degree of natural resistance to the disease. Since none of the control animals died when inoculated with virulent blood, it is difficult to see how any conclusion regarding the proper dose of serum, or its efficacy, can be drawn from the experiments.

I have mentioned the above experiments because, in continuing them, I met with the same obstacle to a definite conclusion, viz., a successful resistance on the part of the control animals, and I believe this difficulty will be met with in all parts of India where rinderpest has established itself in permanence.

It is the general belief that, once a contagious disease has gained a permanent footing in a country, the animals gradually acquire immunity towards it. The reason of this seems to be that the more susceptible individuals succumb year by year, so that the race is propagated mainly by the more resistant, and the progeny partake of the qualities of their forbears. In this sense is the power of resistance hereditary. The resistance is often exalted by a more or less mild attack of the disease which exists in permanence in the country. The degree of hereditary immunity is positive, but seldom absolute.

It seems very unlikely that the mere question of "breed" (in the ordinary acceptance of the term) can have much influence on the degree of immunity. Breed, however, also carries with it an idea of locality, and if it be used in this sense it may be said to have a marked influence on the degree of insusceptibility, for rinderpest has existed in most parts of India for a very long time. (Immunity at certain times seems to be lowered.) It is quite rational to expect that A and B breeds will have a similar degree of immunity towards rinderpest if the disease has for a long time been operating on both. This has been known for years, and required no further experiments to demonstrate it. It is borne out, however, by the experiments performed at Cawnpore, Poona, and Nagpur, in which the animals recovered from the virulent blood inoculation whether they had received serum or not, and it was not surprising to find that the same results attended those experiments (two sets) which I was able to complete in the Madras Presidency before leaving India.

These repeated failures to obtain definite results seem to me to render it inadvisable to continue experimenting on the same lines. What may be expected to happen is that both the serum-injected animals and the controls will take rinderpest and recover when inoculated with virulent blood. Conclusions as to the correct dose of serum can have little value if based on such experiments, more especially when the object is to fix the dose for the serum alone

method. An animal which has received serum may show nothing more than a slight rise of temperature when inoculated with virulent blood, but so may the control which has received no serum, and the influence of natural resistance cannot be deduced from the result, for there is no means of estimating it.

Bearing the former failures in mind, I asked and obtained permission to modify the experiments in the event of the results being unsatisfactory.

The excellent arrangement of hiring instead of purchasing the experimental animals made by Vet.-Major Gunn enabled us to undertake a much more exhaustive scheme of test experiments with the funds at our disposal.

Berhampore Experiments.

Berhampore, Ganjam, was chosen for the first experimental station. Ganjam district returns a large number of deaths from rinderpest, but there is good reason for believing that in some of the apparently more fortunate districts the returns are less carefully attended to.

In order to save time, Vet.-Major Raymond, Supt. C.V.D., Bengal, was asked to co-operate in procuring virulent rinderpest blood, and he was good enough on two occasions to send me the necessary material. I take this opportunity of thanking him for his prompt assistance. In the first instance a modified series of experiments was started, leaving those by the simultaneous method for a later date. The animals to be used for the experiment were kept in kraals. Those used for the up-keep of virulent blood were located four hundred yards distant from the others. They had a special attendant who never went near the other animals. Their drinking buckets were used only for themselves, and their food supply was stored beside the kraal. The water supply was drawn from a source different from that of the others. The veterinary assistants used a special thermometer for taking their temperatures, and they visited the uninfected kraals first. Special shoes were kept for entering the infected kraal, and disinfection of the hands was practised after handling. No case of rinderpest occurred amongst the animals for serum-testing until several days after they had been inoculated with virulent blood.

Effect of Virulent Blood alone on the Local Animals.

On the 20th January virulent blood packed in ice arrived from Calcutta. It had been withdrawn from a sick animal about thirty hours before. On the same day two animals were inoculated—I A, with 5 cc., and 3 A, with 1 cc., for the upkeep of virulent blood. I A showed a slight rise of temperature (39.5 c.) but no symptoms of rinderpest, while 3 A had a severe but non-fatal attack. Blood was withdrawn from 3 A on the fourth day of the disease, and inoculated in the dose of 5 cc. to animal 2 A, which developed a very slight attack of rinderpest. Animal 20 received 5 cc. of blood from 2 A; a slight rise of temperature followed, but no symptoms of rinderpest appeared. Animal 21 received 1 cc. of the same blood with the same negative result. This supply of virulent blood, then, was lost, owing

to the natural insusceptibility of the animals. It was not used for serum-testing, as I thought it would be more satisfactory to obtain a fatal result first. Animals 1 A, 20, and 21 were afterwards housed with and watered from the same trough as twenty others suffering from rinderpest, but they did not take the disease.

A mere trace of virulent blood is sufficient to give rinderpest to a susceptible animal. MM. Nicolle and Adil Bey (*Annales de l'Institut Pasteur*, April 1899) showed that an artificially immunised animal would resist the inoculation of 5000 cc. of virulent blood as successfully as a much smaller dose. The present experiments show that 1 cc. and 5 cc. were equally powerless to give the disease to animals possessing a natural resistance, and had the dose of virulent blood inoculated been 100 cc. the result would probably have been the same. It follows from this that the inoculation of 1 cc. of virulent blood as employed in all the serum-testing experiments in India, is not to be looked upon as a measured test. If the dose of infected blood really mattered, it would be necessary to graduate it according to the weight of the animal in the same way as the serum. In the serum-testing experiments at Berhampore 4 cc. of virulent blood was successfully employed.

The serum alone method is only applicable to herds in which rinderpest exists. A sufficiently large dose of anti-rinderpest serum gives the animals a passive or temporary immunity which gradually passes off. Before it has declined too far, however, it is expected that the fortified animals will be infected from their sick companions, that they will take the disease in a very mild form and recover, and that afterwards they will possess an active degree of immunity.

The duration of the temporary immunity aimed at, then, should be long enough to allow the wave of infection to pass over. With this explanation, the object of the first series of experiments will be apparent.

First Series of Experiments.

On the 21st January nineteen cattle and eight buffaloes were injected with varying doses of anti-rinderpest serum, issued at 7-8 cc. per 600 lbs for plains animals. They were tested sixteen days afterwards with virulent blood. The original intention was to test them on the fourteenth day, but two days were lost through having to obtain blood from Calcutta. The virulent blood, which was sent by Vet.-Major Raymond, had been withdrawn from a bull which subsequently died of typical rinderpest. It had been brought to Berhampore on ice, and was inoculated to the test animals less than thirty hours after withdrawal. The dose employed was 4 cc.

It will be sufficient here to indicate the results as shortly as possible.

Experiments on Mixed Breed of Cattle at Berhampore.

The animals received varying doses of anti-rinderpest Serum XI, issued at 7-8 cc. for plains, and were inoculated sixteen days afterwards with 4 cc. of virulent blood.

No. of Animal.	Age.	Dose of Serum per calculated 600 lbs.	Actual Dose of Serum.	Results.	Remarks.
1	5 yrs.	5 cc.	2 cc.	No symptoms of rinderpest	Highest temp., 39° C.
2	7 "	5 "	2'4 "	do. do.	Highest temp., 39'4° C.
3	5 "	5 "	1'6 "	Very severe attack	Recovery after long convalescence.
4	10 "	6 "	3 "	Slight attack	Recovery.
5	4 "	6 "	2'6 "	Moderately severe attack	do.
6	4 "	6 "	2'4 "	Medium attack	do.
7	2 "	7 "	2 "	Severe attack	do.
8	2 "	7 "	2'4 "	do.	do.
9	2 "	7 "	2'4 "	do.	do.
10	3 "	8 "	2'8 "	Medium attack	do.
11	1½ "	8 "	2'4 "	Severe attack	do.
12	2 "	8 "	2'4 "	Slight attack	do.
13	2 "	9 "	3 "	do.	do.
14	3½ "	9 "	3 "	do.	do.
15	2 "	9 "	3'2 "	do.	do.
16	2 "	10 "	3'2 "	Medium attack	do.
17	3 "	10 "	3'6 "	Slight attack	do.
18	3 "	15 "	5'2 "	Very severe attack	Recovery after long convalescence.
19	5 "	15 "	5'6 "	do.	do. do.
21 A	2 "	Control (none)	None	Slight attack	Recovery.
21 B	2 "	do.	do.	do.	do.
21 C	2 "	do.	do.	do.	do.
21 D	2 "	do.	do.	do.	do.

It is to be noted that the four control animals, which had received no serum, had only a slight attack of rinderpest, and it will be remembered that in four other animals, Nos. 1 A, 2 A, 20, and 21, the degree of natural resistance was even more marked. One would not be justified, then, in crediting the serum with being responsible for the mildness of the attack in the case of Nos. 1, 4, 12, 13, 14, 15, and 17. It is possible that it was due to natural resistance, and the result might have been the same had the animals received no serum at all. This view becomes the more probable when one notes what occurred in the case of Nos. 18 and 19. These animals received the largest dose of all, twice as large as the standard dose at which the serum is issued for the serum alone method, and yet they suffered the most severely of all. So severely did they suffer that the owners stipulated for special compensation before they would take them back a month after the commencement of the disease. It will afterwards be seen that blood drawn from No. 18 was successfully used to produce rinderpest at Ongole. The others were returned at the same time, but in spite of the comparatively generous diet which they had received many had so fallen off in condition that it was found necessary to give the owners compensation. I think it may be fairly concluded from those experiments that serum of the strength of No. XI, given at the dose of 15 cc. per 600 lbs., could not be relied upon to save the more susceptible plains animals from a very severe attack of rinderpest if the infection were delayed sixteen days. It remains

to be seen from further experiments if infection at an earlier date would have much less serious results, but I would point out that in dealing with outbreaks of rinderpest in India no great benefit can be expected from a method of giving immunity which disappears in such a short time. Even supposing that the immunity could be relied upon for, say, ten days, it would mean that every animal which had not had a slight attack of rinderpest by the tenth day would have to receive another dose of serum, and so on until the disease had disappeared from the herd. That, of course, could not be carried out. It might be suggested that in the case of Nos. 18 and 19 the result would have been fatal if they had received no serum at all. On the other hand, however, many animals which have received no serum are known to have recovered from an attack of similar gravity after a prolonged convalescence.

In the case of buffaloes wider intervals were allowed between the proportionate doses of serum. The animals were tested as in the case of the cattle by inoculating 4 cc. of virulent blood sixteen days after the injection of serum. The results were, shortly, as follows:—

Experiments on Buffaloes at Berhampore.

The animals received varying doses of Serum XI, and were tested sixteen days afterwards with 4 cc. of virulent blood.

No. of Animal.	Age.	Dose of Serum per calculated 600 lbs.	Actual Dose of Serum.	Results.	Remarks.
1	2 yrs.	5 cc.	2.2 cc.	Severe attack	Recovery.
2	1½ "	5 "	1.8 "	None	Died four days after inoculation of virulent blood, but not of rinderpest.
3	6 "	8 "	6.6 "	Medium attack	Recovery.
4	3 "	8 "	4.6 "	Very severe attack	do.
5	8 "	11 "	9.2 "	None	Died of an accident.
6	8 "	11 "	11 "	Severe attack	Recovery.
7	2 "	14 "	8.8 "	do.	do.
8	3 "	14 "	10.2 "	Very severe attack	do.
9	2 "	Control (none)	None	Severe attack	do.
10	2 "	do.	do.	do.	do.
11	2½ "	do.	do.	Very severe attack, which did not begin until fifteen days after inoculation—i.e., much later than the ordinary inoculation period.	It is possible that this animal resisted the inoculation in the first case, but subsequently became infected from his fellows.

It will be seen that the results of the experiments on buffaloes were practically the same as those obtained with cattle. All the animals took the disease more or less severely, and, curiously enough, one of them—No. 8—which had received the largest dose of serum

suffered the most severely. None of the control animals succumbed, nor did they, with the exception of No. 11, become more markedly affected than those which had received serum. They all reacted, in fact, as if they had received no protective material at all. No 11 (control) behaved in a curious manner; he resisted the inoculation of virulent blood far beyond the usual incubative period, and then developed the disease in a very severe form, but ultimately recovered. I have suggested that he was possibly infected at the later date by his sick companions with whom he drank and fed. It is to be observed that none of the other buffaloes failed to react distinctly to the virulent blood inoculation, and I may mention that the same held good at Ongole. This animal, then, seems to be a much more trustworthy reactive for rinderpest experiments than plains cattle.

Writing from memory, I believe this has already been pointed out by Vet.-Major Raymond.

The buffaloes were handed back at the same time as the cattle to their owners, who, it was thought, were entitled to compensation on account of the condition of their animals.

Experiments at Ongole.

The object in going to Ongole was to conduct experiments on the Nellore breed (pure breed) of cattle. In order to save time, Vet.-Major Gunn was good enough to make all the arrangements at Ongole, while I was still engaged at Berhampore. The animals collected were for the most part typical of the Nellore breed. One or two, however, had a slight strain of the ordinary plains animal, but this did not appear to make any difference in the way they reacted to rinderpest. The kraal arrangements were the same at Ongole as at Berhampore.

The present method of preparing anti-rinderpest serum is still in the empirical stage, and it was considered useless to attempt to work to a cubic centimetre per body weight, especially as the method of calculating that weight is the reverse of accurate when applied to plains animals in fairly good condition. It was intended also to carry out a series of experiments by the simultaneous method. That intention had to be abandoned, owing to my presence being urgently requested in the Transvaal. In any case, however, it would not have been serviceable to conduct further experiments in the Ongole district, as it was discovered that the disease known as "Red Water" existed amongst the cattle. Although red water appeared amongst the experimental animals, and must be held to vitiate the results of the serum experiments at Ongole, they are not without value, because it will be observed that the control animals also took red water. The conditions, then, being similar in the serum-injected and control animals, a comparison is to a certain extent admissible. Twenty-two cattle were injected with varying doses of Serum XI. on the evening of the 12th February. They were tested, some of a series with 4 cc., and others of the same series with 1 cc. of virulent blood on the morning of the 24th February, that is to say, ten and a half days after the injection of serum. The results are shortly given as follows—

Experiments on Cattle at Ongole.

The animals received varying doses of Serum XI., and were tested with virulent blood ten and a half days afterwards.

No. of Animal.	Age.	Dose of Serum per calculated 600 lbs.	Actual Dose of Serum.	Dose of Virulent Blood.	Results.	Remarks.
22	3 yrs.	5 cc.	3.4 cc.	.4 cc.	Severe attack of rinderpest	Also developed redwater. Recovery.
23	1½ "	5 "	2.8 "	.4 "	Medium attack of rinderpest	Died of redwater twelve days after inoculation.
24	4 "	5 "	3.8 "	.4 "	No symptoms of rinderpest	Highest temp., 40.1° C.
25	4 "	5 "	2.6 "	1.0 "	Severe attack	Recovery.
26	2 "	7 "	4.4 "	.4 "	Mild attack	do.
27	4 "	7 "	3.8 "	.4 "	Severe attack	do.
28	2 "	7 "	2.6 "	1.0 "	do.	Also developed redwater. Recovery.
29	2½ "	7 "	3.8 "	.4 "	do.	do. do.
30	2 "	9 "	4.6 "	.4 "	Medium attack	Died of redwater nine days after inoculation.
31	3 "	9 "	6.6 "	.4 "	No symptoms of rinderpest	Highest temp., 39.6° C.
32	3 "	9 "	7.0 "	1.0 "	do do.	Highest temp., 41.4° C.
33	4 "	9 "	8.0 "	.4 "	Medium attack	Also developed redwater. Recovery.
34	2 "	10 "	5.2 "	.4 "	Very mild attack	Recovery.
35	4 "	10 "	5.8 "	1.0 "	Severe attack	do.
36	3½ "	12 "	9.8 "	.4 "	do.	do.
37	3½ "	12 "	7.2 "	1.0 "	Very mild attack	do.
38	3 "	14 "	10.6 "	1.0 "	Severe attack	do.
39	2 "	15 "	9.0 "	.4 "	do.	Also developed redwater. Recovery.
40	5 "	15 "	10.0 "	.4 "	Mild attack	Recovery.
41	1½ "	15 "	7.0 "	1.0 "	Medium attack	do.
48	1½ "	Control (none)	None	1.0 "	do.	Also developed redwater. Recovery.
49	2½ "	do.	do.	—	Severe attack	do. do.
50	1 "	do.	do.	—	do.	do. do.

In the case of the animals which developed red water an attempt was made to deduce the injurious effects of the latter disease while estimating the severity of the rinderpest attack, but it must be borne in mind that the accuracy of the estimate is open to question.

Two of the controls were severely affected with rinderpest, and one had a medium attack, but all recovered in spite of the red water complication. Some of the animals which had received the higher doses of serum suffered as severely from rinderpest as the controls when infected ten days afterwards. If it be permissible to draw a conclusion from the above experiments, it must be that little benefit can be expected from Serum XI. used in the above doses for the serum alone method, should infection be delayed for ten days. The Berhampore results favour such a conclusion. A preliminary trial was begun with 20 cc. per 600 lbs., calculated weight, but my departure prevented experiments on a larger scale.

On the 21st February two Nellore (cross-bred) cattle, 46 and 47, received 20 cc. per 600 lbs. of Serum XI. They were tested ten days afterwards with 1 cc. of virulent blood. Both animals had a very mild attack of rinderpest, but it must again be pointed out that some

animals which have not received serum may show symptoms of like insignificance, and it would be advisable to have many more results before pronouncing the dose of 20 cc. per 600 lbs. to be efficacious even up to ten days. Had time permitted it was intended to have done a large number of inoculation experiments on cattle which had received 20 and 25 cc. of standardised serum per 600 lbs. using a larger number of controls, and extending the interval between the injection of serum and infection with virulent blood. The experiment, however, which would most nearly approach the natural conditions would be on the following plan:

For each experiment first, say, forty cattle be borrowed from the natives by the arrangement carried out in Madras, which would enable twice as many animals to be used for less outlay than in the case of purchase. Let one half of the animals be injected with the dose of serum under trial, say 25 cc. per 600 lbs., and keep the other half as controls. Immediately after the injection of serum, let two animals in full attack of artificially produced rinderpest be introduced to mix, feed, and drink with the others. One could then carefully observe and note the time taken by the disease to run through the controls and the serum animals. The degree of severity of the symptoms in the two sets of animals would form an interesting and useful comparison. If it turned out that the symptoms of rinderpest were decidedly more marked in the unprotected animals than in those injected with serum, that none of the latter developed the disease in a severe form, and that the induced ezootic lasted long enough to be at least comparable with an ordinary outbreak in a herd, one would be within measurable distance of arriving at the correct dose. One could even lengthen out the experiment if necessary, by introducing fresh cases. The material already obtained in practice (from outbreaks) cannot be looked on as satisfactory evidence of this kind.

A comparison of the severity of the disease, for example, can seldom be made in animals of the same herd which have received serum, and those which have not. In reports of outbreaks we find simply the death rate before serum treatment was commenced, compared with the number of fatal attacks after it was resorted to. Nothing is said of the severe cases which do not end in death, and in many outbreaks these outnumber the fatal ones, even if serum be not given. If we pronounce on the efficacy of the dose then, by comparing the death rate in an outbreak before and after serum treatment, we are likely to confuse post hoc with propter hoc.

If, as often happens, the inoculator operates on a herd some considerable time after the commencement of the outbreak he deals with a herd from which many of the most susceptible individuals have been eliminated and the disease may have practically worked itself out. A number of deaths, however, are recorded after serum treatment has been resorted to, and the explanation offered is that the animals had contracted the disease prior to receiving the serum. This, of course, is quite conceivable, but the other important possibilities have apparently never been considered. These are:—

- (a) That the fatalities occurred (at an early stage) amongst the more susceptible individuals—the very animals we aim at protecting—owing to the dose of serum being too small.
- (b) That whatever protection was at first derived from the dose of

serum employed had completely passed off before the outbreak declined, and in consequence fatal cases occurred amongst susceptible animals in which infection had been delayed.

The question is important, and one part of it at least admits of a fairly satisfactory settlement. The incubation period after inoculation is not less than three days, although it may be longer. By the natural method of infection it is from three to ten days, but the extremes are unusual. No case of rinderpest, then, can be fairly said to be due to infection previous to the operation unless evidence is brought to show that the disease commenced as early as the third day after injection of serum. This evidence has not been stated in support of the assertion that the fatal cases after serum infection were due to the animals being already infected, and it seems more likely that they were due to an insufficient dose of serum.

Attempts to Overcome the Natural Resistance.

When one experiments on a plains animal one cannot determine the part played in the result by natural resistance. Were it possible, by means of reagents or toxins, to reduce the natural resistance of plains animals to the level of the susceptible class (hills, for example), so that inoculation with virulent blood would almost certainly produce a fatal result, one would be in a position to measure with reasonable accuracy the difference of susceptibility between this and that animal. The resistance of the individual plains animal varies so greatly that it is hardly possible to compare it as a whole with that of another class. In estimating the dose of serum, however, this would not matter if we could determine the relative resistance of the more susceptible of the plains animals. (It is these we desire to protect.)

In the case of the disease known in England as "black leg," solutions of acid and alkalies have been successfully used to overcome the natural resistance of certain animals. Cultures of non-pathogenic bacteria have been similarly employed.

I performed a few preliminary experiments with lactic acid and glycerine in relation to rinderpest, but the results were irregular and inconclusive.

Lactic acid in doses of 1 cc. was injected into nine animals which had been inoculated with infective blood. Three took the disease severely, one had a medium attack, and in five the disease was mild.

Seven buffaloes were inoculated with 1 cc. of virulent blood, and glycerine was also injected into five of them, in doses varying from 8 to 40 cc.; two were kept as controls. One animal which had received 15 cc. of glycerine died of rinderpest. All the others were severely affected, but not in proportion to the amount of glycerine injected. The two control animals also took the disease severely, but they recovered.

One cannot extract much from these few preliminary experiments, and time did not permit me to continue on the same lines. The advantage to be gained, however, from being able to fatally infect without fail a plains animal with rinderpest would be so great that I think the question is worthy of further investigation.

In the case of Serum XI., I find the statement that the breeds of the plains are eighteen times less susceptible than hill breeds open to

serious question, nor can I in view of the varying susceptibility of plains animals see what correct data one possesses to enable one to make such a statement.

Even were it correct, however, it could have no practical value, for the important thing to aim at in India is the protection of the most susceptible, rather than of the average individual animal which would recover from rinderpest without the aid of serum.

In South Africa, where serum treatment has rendered invaluable service, the standard of 10 cc. per 600 lbs. body weight was chosen because 600 lbs. was approximately the average weight of the cattle. A minimum dose, however, was recognised, and one did not fall into the absurdity of giving an animal 2, 3, or 4 cc. Such a proceeding appears to me to be a waste of a rather expensive material.

One might with advantage in India try a minimum dose of 15 cc., irrespective of body weight, for animals directly exposed to infection. For the protection of those outside the immediate area of infection a longer period of immunity would be required, as the infection would take longer to spread. The dose of serum in this case would require to be much larger.

The method in vogue in India of calculating the dose of serum for the serum alone method is based on a misconception of what the serum is intended to do in practice. With a normally susceptible animal (the plains animal is too uncertain for a standard) experiments by the simultaneous method only fixes the dose of serum which will protect against immediate infection.

An animal affected with rinderpest, however, may be capable of infecting its fellows for a fortnight or more, and it must be apparent that unless the dose of serum is capable of giving immunity for this period it can be of little use in dealing with rinderpest in India, where the natives do not understand sanitary police.

REPORT REGARDING EXPERIMENTS ON CATTLE WITH TUBERCLE BACILLI DERIVED FROM VARIOUS SOURCES.¹

By Regierungsrath, Prof. Dr H. KOSSEL.

IN compliance with a request from our President, I intend this evening shortly to recount the results we have hitherto obtained in the Imperial Hygienic Department from our experiments on oxen with tubercle bacilli derived from various sources.

The experiments were chiefly intended to solve the question whether cultures of tubercle bacilli obtained from man, would produce the same pathological effects on cattle as those obtained from tuberculous lesions in cattle and swine.

Even before his well-known lecture at the British Tuberculosis Congress in 1901, Robert Koch had described before an assembly of experts in the Sanitary Institute the results which he had obtained in conjunction with Herr Schütz, and had expressed the wish that a Commission might be appointed which would still further test this question.

¹ Translated from the "Berliner Klinische Wochenschrift," No. 29, 20th July 1903, p. 653.

In consequence, a considerable sum of money was voted for this purpose in the next session of the German Parliament, for it was foreseen that the experiments would be somewhat costly and of long duration.

On the 2nd April 1902 the Commission of the Imperial Sanitary Board for the investigation of tuberculosis met and formulated a plan according to which the experiments on animals should be carried out. As Director of the Bacteriological Laboratory of the Sanitary Institute I was entrusted with the task of carrying out these experiments. I had the advantage of having associated with me Herr Stabsarzt Dr Weber, and Rossarzt Dr Heuss. During the past year Dr Weber has carried out very extensive and important work. Of this I can only give an idea when I say that more than one hundred different varieties of tubercle bacilli have been cultivated, and the greater part have afterwards been tested in regard to their pathological action on cattle.

The fundamental principles on which the plan of experiments rested were as follows. It was thought advisable to avoid working with material directly derived from dead bodies, and first of all to prepare pure cultures of the tubercle bacilli, the pathological action of which on cattle should then be tested by the method of subcutaneous injection. This determination was arrived at on account of the fact that, as received in the Laboratory, the material from dead bodies is usually associated with other bacilli, and it is well known that the presence of foreign organisms may exercise an important influence on the course of a given infection, and that bacteria which of themselves are absolutely incapable of producing disease may exhibit pathological action when accompanied by other micro-organisms. It was also considered necessary to use as far as possible equal quantities of the infecting matter. The material obtained from dead bodies was therefore utilised for infecting guinea-pigs, from which again cultures were made on serum, and from bacilli thus isolated mass cultivations were prepared on bouillon. After growth on bouillon had proceeded sufficiently far the film formed of bacilli was collected, weighed, mixed with a given quantity of fluid, and rubbed down in a mortar, and a similar amount of tubercle bacilli, viz., 5 centigrammes, was used for the injection of each subject.

The plan of experiments was conceived with the idea of also throwing light on other points, such as whether cattle could be infected by feeding on sputum from phthisical human patients and tubercle bacilli of human origin, or by being caused to inspire these bacilli. The two latter points have not yet been entered upon. I shall therefore confine myself this evening to considering shortly the first point, viz., whether pure cultures obtained from cases of tuberculosis in man can by subcutaneous injection into cattle produce progressive tuberculosis, like the tubercle bacilli of the ox, as shown by Koch and Schütz.

To begin with I shall shortly sketch the symptoms which follow subcutaneous injection of cultures of the bacilli of perlsucht or of porcine tuberculosis into the ox. After some days a swelling begins to develop at the point of inoculation. At first it is somewhat diffuse and doughy, and extends in all directions, covering an area as large as a cheese plate. During the course of the next few days it markedly

increases. In eight to ten days the neighbouring lymphatic glands become enlarged. When the bacilli of perlsucht or of porcine tuberculosis have been injected these glands during the next few weeks may attain the size of a child's head. Infiltration at the point of inoculation and enlargement of the prescapular lymphatic glands usually remain well marked until death, or until the experiment is brought to an end by slaughtering the animal.

Furthermore, in about eight to ten days after injection the animals show high fever, which, in fatal cases, continues until death. In other cases (in which death does not occur) the temperature may, however, again gradually sink. The temperature curves are irregular, or, as in one case, may sink to normal.

We now possess several varieties of tubercle bacilli derived from perlsucht in cattle and tuberculosis in swine, all of which, as Robert Koch described, are capable of rapidly extending from the point of inoculation to the internal organs of the animal, and of producing death by generalised tuberculosis. When one makes a *post-mortem* examination of an animal which has died in eight to nine weeks after receiving a subcutaneous injection of this kind, one finds at the point of inoculation an extensive caseous infiltrate, the centre of which may have undergone softening, or which may even reveal extensive calcareous deposits. The prescapular glands, as I have previously mentioned, are enormously enlarged. Under any circumstances, they are either entirely and equally caseated, or exhibit closely-packed caseous centres. Numerous calcareous concretions are also present. The spleen shows numerous tubercles. The lungs are crammed with tubercles varying in size between a grain of linseed and a pea. The kidneys, liver, and most of the lymph glands of the body contain tuberculous centres; in a word, these cases show all the signs of generalised tuberculosis.

The infection with perlsucht bacilli does not always follow an acutely fatal course. The original infiltration may gradually be absorbed, and the prescapular glands diminish in size, though very considerable swelling always remains at both points. In such cases it becomes necessary to slaughter the animal, and this we have usually done after about four months. In such animals we also find generalised tuberculosis of the whole body.

Altogether we have tested seven cultures of tuberculosis from cattle and swine—four from oxen and three from swine. Two strains killed cattle with acute symptoms in eight to nine weeks. The other four cultures likewise produced generalised tuberculosis, which, however, followed a more chronic course. Of these seven cultures, only one failed to produce the results above sketched when injected under the skin. An infiltration at the point of inoculation certainly occurred, and caseous centres developed in the nearest prescapular gland, while similar centres were found in one of the mediastinal glands, but beyond this there was no indication of the extension of tuberculosis throughout the body, such as we usually found after injection of cultures of bovine tuberculosis. The virulence of bovine tubercle bacilli may therefore vary to a considerable extent. Speaking generally, however, we may say that, of seven cultures, six appeared capable of producing exceptionally severe and extensive tuberculosis in cattle.

We have now to consider the results obtained with cultures originally derived from human tuberculous organs. Through the kind assistance of our President and of Professors Langerhans and Baginsky, we were enabled to utilise for experiment various forms of human tuberculosis.

First of all we prepared cultivations of tubercle bacilli from nineteen cases of tuberculosis of the lung. The tubercle bacilli were either cultivated from sputum or from organs obtained on *post-mortem* examination. We endeavoured to obtain severe cases of tuberculosis of the lung, *i.e.*, such as showed secondary symptoms in connection with the bowel, and, if not generalised miliary tuberculosis, at least extension of the process to other parts of the body; cases, in fact, which by their progress and by the symptoms which they presented on *post-mortem* examination produced the impression of having been severe or acute. Nevertheless, with these nineteen cultures we obtained results entirely different from those given by cultures of bovine tuberculosis.

When a culture obtained from a phthisical human subject is subcutaneously injected into an ox there occurs during the next few days a certain amount of infiltration at the point of inoculation. In from eight to fourteen days the prescapular glands also show swelling, but in most cases no rise of temperature whatever is noticeable. The swelling at the point of inoculation and that of the prescapular glands, however, never attains the size seen when cultures of bovine tuberculosis have been employed. These glands never become larger than a goose's egg, and after attaining this size they begin to diminish. If after about four weeks the animal be killed, one finds in a large number of cases that the remainder of the injected tubercle bacilli are still traceable at the point of inoculation in the form of encapsuled abscesses containing caseous material. The tubercle bacilli are still living, as proved by the inoculation of experimental subjects, but comparatively few can be found. The prescapular glands, though at first swollen, have usually returned to their original size, and display no signs of the morbid processes from which they have suffered.

In a small number of cases, however, one finds in these glands caseous centres, some as large as peas, or even as beans, four months after injection. Extensive tracts of connective tissue in the neighbourhood of these centres show, however, that the system has been successful in its attempts to limit the extension of the tuberculous process. In other cases, again, we find in the prescapular glands tuberculous centres which do not exhibit such marked encapsulation, but in which small tuberculous points have extended from a large centre (perhaps the size of a hazel nut) into the surrounding gland tissue. These centres, like those previously referred to, occasionally exhibit calcareous degeneration, but in no single instance among these nineteen cases injected with cultures of human phthisical material have we discovered that extension of tuberculous processes to the internal organs of the ox which (with the single exception mentioned) was the rule when employing cultures obtained from bovine *perlsucht* and porcine tuberculous material.

If one regards these results obtained with tuberculosis of the lung by themselves, one is at once obliged to accept Koch's view, *viz.*,

that a difference exists in the pathogenic power of human tubercle bacilli and those of the ox as correct.

Koch, however, has already directed attention to the fact that cases of human tuberculosis may occur as a result of infection with *perlsucht* bacilli. He himself therefore agreed that as large a number as possible of different varieties of human tuberculosis should be tested, in order to see whether amongst these different forms variations might not be detected.

Regarding the most important of these forms, tuberculosis of the bowel, Koch, in 1901, spoke as follows:—

"I could cite from the literature of the subject a considerable number of cases of that kind, which undoubtedly show that primary tuberculosis of the bowel, particularly in children, is a relatively rare disease, and of those few cases which have been collected not one has been proved due to infection by bovine tuberculosis. It might just as well be human tuberculosis, which is widely distributed, and may gain entrance to the digestive canal in one of many ways, *e.g.*, by swallowing saliva infected with human tubercle bacilli through the mouth. Up till now no one has been able to declare with certainty whether the cases of tuberculosis of the bowel hitherto discovered were of human or animal origin. Now, however, we can establish the diagnosis. It is only necessary to prepare from the suspected material pure cultivations of the tubercle bacilli, and with them to inoculate cattle, in order to prove whether or not they are derived from bovine sources. For this purpose I recommend subcutaneous injection, which produces extraordinarily characteristic and convincing results."

The plan of experiments was drawn up in accordance with this proposal, and in addition to cultures of tuberculosis of the bowel we have tested a large number of tubercloses derived from other sources in regard to their pathogenic properties for the ox.

I shall first describe the action of cultures from tuberculosis of bone. Of this variety of tuberculosis, we examined four cases—three from adults and one from a child. Some of these cases exhibited extensive and severe disease of the osseous system. Of four cultures thus obtained only one proved capable of producing disease, which consisted in the appearance of caseous centres in the prescapular glands.

When the affected animal was killed, after a period of four months under observation, we found two caseous, partly calcified, centres as large as walnuts in the prescapular glands on the side inoculated, but no generalised tuberculosis. None of the three other cultures proved capable of producing generalised tuberculosis in cattle. The injections simply produced infiltration and temporary swelling of the prescapular glands.

We then tested two cultures from cases of tuberculosis of the cervical glands in children. Both cases showed the above-mentioned disease of the prescapular glands, which, however, exhibited no markedly progressive character, but was somewhat more serious than that observed in most cases after the injection of pulmonary tuberculous material. The cattle, however, did not show any generalised disease.

Injection of a culture of uro-genital tuberculosis produced similar results.

We now come to a group of tubercloses of particular interest, viz., the miliary tubercloses. Up to the present time I have succeeded in obtaining six cases of this kind—five in children and one in an adult. In several cases it was evident that the disease had originated in the digestive tract.

To describe these cultures somewhat more closely, I may say that one was obtained from a case of miliary tuberculosis which had succeeded disease of the bowel. The child showed ulcers of the bowel, tuberculosis of the cervical lymph glands, tuberculosis of the tonsils, and generalised miliary tuberculosis. Cultures obtained from the tonsils were, however, practically without effect on oxen. After four months only a little yellow centre the size of a peppercorn could be found in the prescapular gland of the side injected, and this could easily be shelled out and showed no trace of calcification or suggestion of an invading character. A culture from an eight-and-a-half-years-old child which had died of miliary tuberculosis, and on *post-mortem* examination showed caseation of the mesenteric glands, swelling of the retro-peritoneal glands, and tuberculosis of the mediastinal and cervical glands, behaved in a similar way. The effect on the ox was practically nil.

Two other cases closely resembled these. One was that of a twenty-six-years-old man suffering from pulmonary tuberculosis and syphilis in addition to miliary tuberculosis. The other was a six-months-old child which showed caseous pneumonic centres in the lung, caseated bronchial and tracheal glands, miliary tuberculosis of the spleen, liver, and kidneys, tuberculosis of the bowel, caseated mesenteric glands, and small tubercles, the size of grains of linseed, in the brain.

From the cases hitherto described, however, two, which were also obtained from cases of miliary tuberculosis in man, must be distinguished. One of these consisted of generalised tuberculosis with caseation of the bronchial glands. Unfortunately we could not discover whether the mesenteric glands were also diseased. The injection of this culture into an ox produced more serious symptoms than we had previously seen in consequence of the injection of bacilli of human origin. We repeatedly made subcutaneous injections of the culture into oxen, employing more than four animals in all. In these subjects the point of inoculation was marked by more pronounced swelling than we had ever before seen, and this swelling did not retrocede. The prescapular glands were also enlarged to a greater extent. They attained the size of a man's clenched fist and remained so. Nevertheless, the animals scarcely showed any fever, only a slight irregularity of temperature, and the temperature curve did not nearly resemble that obtained after the injection of *perlsucht* bacilli. The subjects were killed after about 125 days, and, in addition to the changes at the point of injection and in the neighbouring lymphatic glands, we found generally distributed tuberculosis of the entire body, including tuberculosis of the spleen, the lungs, the liver, and the kidneys. The general appearance reminded us strongly of that seen after injection of *perlsucht* bacilli; not perhaps of the acutest type that we had then observed, but of the cases which had followed a somewhat more chronic course, and which, without rapidly killing the animals, had nevertheless produced widely disseminated tuberculosis.

A second culture obtained from the caseated mesenteric gland of a three-and-a-half-years-old child behaved in a similar way. The child showed the appearances of miliary tuberculosis and of tuberculous meningitis. The animal injected with this culture showed, after 122 days, extension of the tuberculous processes to the internal organs, in addition to the well-known symptoms at the point of inoculation and in the prescapular glands. Intravenous injection of the same culture into an ox caused death with characteristic symptoms in about twenty days.

We now come to the cases of primary tuberculosis of the digestive tract. Seven such cases in all were examined. Three occurred in adults and four in children.

Dealing first with the three adults, one showed a pure tuberculosis of the bowel, *i.e.* the *post-mortem* examination revealed only a single tuberculous ulcer in an inguinal hernia. The patient, who was seventy-seven years of age, had died of strangulated hernia. During life no one had suspected the existence of tuberculosis. This fact rendered it probable that the disease had not long existed.

The view has repeatedly been expressed that bovine tubercle bacilli might lose their virulence for oxen in consequence of lengthened retention in the human body. As the case now under consideration was probably recent, we might naturally have expected the culture to be of marked virulence; nevertheless, an ox inoculated with it remained completely healthy after 136 days, except for the existence in one of the prescapular glands of a few minute abscesses, varying in size between a grain of linseed and a pea, and containing tubercle bacilli.

A culture from the body of an adult woman who had died from tuberculosis of the bowel and perforating peritonitis, and in whom tubal tuberculosis was also detected, produced somewhat more strongly marked symptoms. After 118 days the prescapular gland of the inoculated ox was found to contain a number of caseous centres, some as large as a hazel nut, and a caseous centre in the middle cervical gland on the infected side. On the other hand, the other organs of the animal were entirely free from any suggestion of tuberculosis referable to the inoculation. The investigation of these cultures is, however, not yet completed, though it appears that they should rather be included in the group which is not pathogenic for the ox.

Another culture, obtained from a fifty-eight-years-old man who had died from peritonitis, and in whom a large bowel ulcer and swelling of the mesenteric glands was found on *post-mortem* examination, was discovered to be non-pathogenic for the ox.

In regard to the four children with primary tuberculosis of the digestive tract, the results of the investigation are of great interest. I will first describe the results obtained with a culture from a child which on *post-mortem* examination showed tuberculosis of the bowel and calcification of the mesenteric glands. At the present time, after 112 days, the ox is still healthy, and judging by previous experience we can well believe that we shall find no marked symptoms after killing the animal.¹

¹ The ox has since been slaughtered. It was free from tuberculosis.

This culture must therefore also be included in the group of human tubercloses. The results were also negative in the case of material obtained from tuberculosis of the bowel and caseated mesenteric glands in a two-years-old child which had died of pneumo-thorax. These tubercle bacilli also were incapable of causing any marked disease in cattle.

It should be particularly noted that from cases of apparently primary tuberculosis of the bowel, cultures may be prepared which show the qualities, not of bovine but of human tuberculosis, a fact which gives no support to the view that such cases have been produced by bovine tubercle bacilli swallowed with the food.

On the other hand, this group of cases of tuberculosis of the bowel includes two instances in which the cultures exhibited a particularly well marked pathogenic character for cattle. One case was that of a child which had died from septic scarlatina, and in which the *post-mortem* revealed the existence of caseated and calcified mesenteric glands as a secondary condition. The culture from this case was recently used to inoculate an ox. The symptoms which have already developed showed that it is pathogenic for oxen. The same is true of a culture obtained from the caseated mesenteric glands of a five-and-a-half-years-old child. The child, which had died from peritonitis and fibrinous pleurisy, showed a trifling growth of miliary points on the pleura of one side and a tubercle in the capsule of the spleen, but no other tuberculous changes. The culture when intravenously injected killed oxen in a short time. When subcutaneously injected it produced similar symptoms to cultures of bovine tuberculosis.

Summing up the results, I may remind you that we have tested thirty-nine different cultures freshly prepared from tuberculous lesions in man, that twenty-three of these cultures were from adults and sixteen from children, and that nineteen of these failed to produce the slightest symptoms in oxen. Nine cattle after the lapse of four months showed very trifling centres in the prescapular glands, such centres being for the most part encapsuled, and certainly exhibiting no tendency to extension. In seven cases the prescapular glands were somewhat more extensively diseased, but even here the process had not really extended beyond that point. Of the thirty-nine cultures, four (two of which were primary tuberculosis of the digestive tract and two of miliary tuberculosis in children) produced generalised tuberculosis in the ox. As already stated, these cultures were not so virulent as the most virulent cultures of animal tuberculosis. They behaved, in fact, more like the weaker forms of bovine tuberculosis.

Two of the children from whose organs the cultures which proved pathogenic for cattle were obtained died from causes other than tuberculosis. In one the tuberculous process was represented by an almost completely calcified centre; in the other, death was due to an inter-current disease.

On the other hand, a great number of human tubercloses, by far the greater portion, yielded cultures which did not produce the impression of having originally sprung from oxen.

Of the two other cases in children one might say that death had resulted from infection with tubercle bacilli which were just as pathogenic for cattle as are many *perlsucht* bacilli. The results of these

examinations are therefore in harmony with those of other investigators, whose work, on account of the short time at my disposal this evening, I cannot individually refer to.

If now we consider the extreme rareness of primary tuberculosis of the bowel, and remember that according to our experiments a great part of this rare disease cannot apparently be referred to infection with tubercle bacilli of bovine origin, but must have resulted from the reception of tubercle bacilli probably rejected from the lungs of diseased human beings, you must allow that Koch was justified in his view that the consumption of materials (like milk, meat, etc.) from tuberculous animals does not play that part in producing tuberculosis in man which has on many sides been attributed to it. On the other hand, we are compelled to say that if Koch's view is correct, viz., that only *perlsucht* bacilli are capable of producing extensive progressive tuberculosis in oxen, we must admit that the children of whom we have just been speaking were killed by *perlsucht* bacilli. In such a case we cannot regard the consumption of uncooked milk obtained from animals affected with *perlsucht*, and of uncooked food products prepared from such milk, as entirely innocuous.

It also appears possible, however, that the bacilli of human tuberculosis may under certain conditions attain a very marked pathogenic character for cattle, without themselves being *perlsucht* bacilli. It therefore becomes our task to seek for further differentiating characteristics. We must also continue the study of allied questions relating to the ingestion and inhalation of cultures of tubercle bacilli derived from the most varied forms of human and bovine tuberculosis, in order to discover whether or not differences really exist. Should these experiments not lead to a unanimous conclusion, we must return to clinical, pathological, anatomical, and statistical observations. Under any circumstances it is necessary to continue collecting clinical and statistical material, in order to decide whether and how often men become infected with tuberculosis in consequence of consuming food products obtained from tuberculous animals.

THE PRESENT POSITION OF THE DISPUTE REGARDING TUBERCULOSIS.¹

By J. ORTH, Berlin.

PERLSUCHT AND HUMAN TUBERCULOSES.

THE question of the communicability of tuberculosis from animals to man is of such importance as to demand the widest possible discussion and the most careful weighing of arguments, whether in one direction or the other.

The main question resolves itself into two secondary questions, which must receive separate consideration, because they may be answered in entirely different senses, viz., the general scientific question whether animal tuberculosis can be conveyed to man, and the special practical question of how great is the danger in any given case that animal tuberculosis will be so conveyed. Should the general

¹ Translated from the "Berliner Klinische Wochenschrift," No. 29, 20th July 1903, p. 657.

question be answered in the negative, should it be proved that animal tuberculosis cannot possibly be conveyed to men, the special question regarding the extent of the danger falls to the ground. On the other hand, however, the first question may very well be answered in the affirmative, and the possibility of the conveyance of tuberculosis from animals to men may be proved without the second question necessarily being positively settled, *i.e.*, without the degree of danger being exactly assessed.

It is also important to keep these two secondary questions apart when considering the converse of the chief question, *viz.*, whether human tuberculosis can be conveyed to animals, because the point of departure of the general discussion which has occupied the two last years, Koch's address to the London Congress, contained, not the contention that the conveyance of tuberculosis from men to animals was difficult and happened but rarely, but the general and dogmatic statement that human tuberculosis cannot be conveyed to animals. Here, then, the general question of the possibility of conveyance is at once denied, a fact which ought carefully to be kept in mind in judging of the present position of the question. The question whether animal tuberculosis can be conveyed to men can only be answered with certainty by observations on men. The publication of cases bearing on this point, like those recently reported by Herr Lassar, are therefore to be welcomed. Care is certainly required in forming conclusions, and I gladly recognise the service of R. Koch in sharply criticising the interpretation of known facts, and in showing how little of our knowledge is certain and unassailable. At the previous meeting Herr Schütz also advanced grounds for believing, not that *perlsucht* could not be conveyed to men (he, like Herr Koch, had admitted the possibility of its conveyance at the tuberculosis Congress), but that infection with *perlsucht* played no considerable part in producing progressive tuberculosis in man.

Herr Schütz added to the cases of tuberculosis of the skin reported by Herr Lassar, and emphasised how much oftener the so-called anatomist's or "dead-body tuberculosis"¹ occurs in men who handle human bodies than in those who deal with the bodies of animals. This was advanced to show how much more easily men became infected with human than with animal tuberculosis, *i.e.*, how differently human and animal tuberculosis behaved. In connection with this matter, I might point out that human tuberculosis is much more extensively distributed than the same disease in animals, and particularly, that the so-called "open" tuberculosis, *i.e.*, changes in which bacilli are discharged from the surface, are very much commoner in men than in animals. Anyone who has noticed how medical men, who frequently possess trifling injuries on their fingers, carry out the *post-mortem* examinations of phthisical corpses exhibiting cavernous spaces crammed with bacilli in the lungs, must certainly wonder at the infrequency of bad results, and must be convinced that tubercle bacilli penetrate the skin only with the greatest difficulty. Nevertheless, these facts go to prove that infection may more readily occur with human material. I leave on one side the question whether one

¹ The German term is *Leichentuberculose*, *i.e.*, "corpse tuberculosis." The term is applied to localised tuberculous due to infection while dissecting or *post-morteming* tuberculous subjects (Transl.).

should regard as really tuberculous all tuberculous appearances found on *post-mortem* examination, particularly that seen on dissecting tables, because it certainly appears to me remarkable that the preparation of muscles, vessels, and nerves, *i.e.*, of parts which only in exceptional cases contain tubercle bacilli, should so frequently result in tuberculous infection. But on one point there can in any case be no doubt, viz., that "dead-body tuberculosis" results in the greater number of instances from mixed infection, and in this connection it is worthy of consideration whether the greater frequency of "dead-body tuberculosis" in those who have to do with human bodies is not explained by the greater liability in their case to septic diseases. Whereas animals are slaughtered and cut up whilst still in a fresh state, human bodies are only dissected long after death, and in almost every case the subjects have died of disease. In handling corpses the chance therefore of septic infection must be very much greater than in dealing with the bodies of animals.

Adopting, however, for the moment, the standpoint of Professor Schütz, I am forced to express my surprise that that gentleman, in order to show the harmless character of the dermatitis tuberculosa produced by perlsucht material, has compared it with lupus of man, and not with the "dead body tuberculosis" produced by handling corpses to which he himself had drawn attention. Is there in reality an essential difference in the severity of the disease? Do not many cases of "dead body tuberculosis" recover spontaneously, do not many pathologists carry lesions of this dead body tuberculosis on their hands for years without the disease making any appreciable progress, are not the cases of this "dead body tuberculosis" which eventually become complicated with changes in the lymphatic glands extremely rare? In my opinion there is no foundation for the view that this "dead body tuberculosis" in man constitutes a much more virulent form of tuberculosis than that contracted from animals, and I cannot therefore recognise the benign character of the latter disease as a sufficient ground for establishing an essential difference between human and animal tuberculosis.

The attempt of Professor Schütz to prove such a difference on the ground of the varying morphological appearances in tuberculous men and animals is no more convincing. When shall we cease to be troubled with this argument, whose meaningless character has so often been exposed. Is it then really so difficult to understand that men are not oxen, and that a grass-eating ruminant will react otherwise, nay, *must* react otherwise, to a given cause than a man? Let anyone mention a single microparasite which produces in all animals exactly the same morphological changes. Such an one does not exist, and why should we expect that the tubercle bacillus should constitute the solitary exception. When a guinea-pig or a rabbit is inoculated with human tubercle bacilli, does tuberculosis develop exactly as in man? Has it not required much pains to dissipate the idea that only miliary tuberculosis can be produced in these animals. Let anyone examine the four preparations from calves infected with human tubercle bacilli here presented; do not they show changes with the characters of perlsucht—the richness in giant cells of the fresh granulations, and the rapid calcification of the caseated areas?

From what has already been said then it follows that the specimens of *pia mater* and omentum which Herr Schütz brought forward do not prove the specific organisms to be different in men and animals—even supposing them to be correct; they are, however, not correct! Herr Schütz has apparently never seen a case of chronic tuberculous meningitis in man, he only knows the acute forms; nevertheless a chronic form also occurs, distinguished by extensive formation of granulation tissue, in which neither the formation of giant cells nor caseation is absent. I have there set out for your examination a preparation of this kind, a section which shows both caseation and the presence of several large giant cells.

Still less apposite is Herr Schütz's example of omentum, for whilst chronic tuberculous meningitis is at any rate rare, that form of omental tuberculosis in which the fatty tissue of the omentum exhibits diffuse formation of granulation tissue, with interspersed caseated centres and tubercles containing giant cells, is one of the commonest of all conditions in somewhat old-standing cases of tuberculous peritonitis. In such cases masses of bacilli are often found around the blood vessels, and I can most strongly recommend all those who wish to study the process by which bacilli make their way into open vessels to carefully examine this omentum, which has become shrunken and converted into a thick cord or into a mass resembling wood. A microscopical specimen cut from a frozen specimen which I have recently shown to my hearers in the demonstration course is displayed there under a low power; please to move the specimen and to note how in certain places the giant cells are collected in great numbers. Another section from an earlier but similar case shows in the caseated neighbourhood a little open vein, whose endothelium has partly been loosened, and into the lumen of which red-stained tubercle bacilli can clearly be traced.

Gentlemen, I fully recognise the conviction with which my teacher Virchow entered into this question of tuberculosis, and, so far as his views on the multiplicity of morphological changes is concerned, I have done my best to give the matter unprejudiced consideration. I therefore have less compunction in emphasising my opinion that Virchow's view regarding the essential nature of the tubercle itself, *i.e.* that tuberculosis cannot exist without the actual existence of a tubercle, can at this date no longer be regarded as justified. Look carefully, gentlemen, at the preparation of a tuberculous tendon sheath shown under that microscope—I have selected it because Herr Schütz spoke of such a case—and you will see a uniformly distributed granulation tissue from which some epithelioid-celled tubercles with unusually large giant cells stand out in consequence of their less marked staining; and then look at the next specimen from a tuberculous ureter. There is no trace of tubercle (Knötchen) formation, and no giant cells to be seen, but only a uniform granulation tissue with caseation of the superficial layers; and this preparation, which is absolutely typical for this particular organ, was taken from a case of chronic tuberculosis of the kidney—a phthisis renalis tuberculosa which swarmed, so to speak, with tubercle bacilli. In man then the infectious disease termed tuberculosis may be accompanied by the formation of diffuse granulation tissue with or without tubercles, but not without tubercle bacilli. Just as its macroscopic appearances vary, so do the microscopic appear-

ances of tuberculosis or phthisis present the most varying picture, and nothing at present recognised either macroscopically or microscopically in animals or in men justifies an attempt to establish an ætiological difference between human and animal tuberculosis.

Koch's own criticism of the facts supporting the view that *perlsucht* can be conveyed to men has shown, in my opinion, that unassailable observations on men are scarcely to be looked for, for further points will always be discovered which will give rise to further doubts.

Exact investigation will then always be confined to the experimental path; and, as in general there is no direct way available, it will become necessary to invert the question and to ask: Is human tuberculosis transmissible to animals? Certainly this question has a certain significance in and for itself, an economic interest, but that point we pathologists and medical men may leave to the veterinary surgeons; for us the great significance of this question lies in the fact that it is so intimately associated with that other, viz., whether animal tuberculosis can be conveyed to men. The one does not necessarily imply the other, but if it can be shown that tubercle bacilli from a severe case of human tuberculosis when inoculated into cattle also produce tuberculous changes, then either genuine human tuberculosis must be capable of conveyance to animals, or, if the human being was not suffering from genuine human tuberculosis, but from animal disease conveyed to him, it is equally evident that animal tuberculosis occurs in man, not as a trifling local tuberculosis but as a severe general disease. Whichever be the fact, in either case the closest possible relationships must exist between human and animal tuberculosis, and in both cases public hygiene demands that public advisers should observe the greatest caution.

What is the present position of the question regarding the conveyance of a human tuberculosis to animals? Herr Schütz had given the Society to expect that he would report concerning the well-known negative experiments which have furnished the basis for Koch's declaration "that human tuberculosis is not transmissible to cattle"; he has, however, finally foreborne to do so in view of the recent reports of the Imperial Hygienic Department, and has submitted himself from the outset to the distinction therein contained. From the scientific standpoint we cannot accept the view that "*Roma locuta est, causa finita*" (Rome has spoken, the case is closed); on the contrary, every one, be he what he may, ought to reserve his powers of weighing evidence free and untrammelled, for here we have to deal with a momentous expression of opinion, momentous because of the origin of the experiments, momentous on account of their number. A week ago, therefore, I willingly abstained from entering into the discussion, the more so inasmuch as I was firmly convinced that the results of the Imperial Hygienic Department would not differ from those of the majority of investigators. So it has proved, and to-day we can say with even greater certainty that the conclusions which Herrs Koch and Schütz drew from their negative experiments, and which culminated in the declaration that human tuberculosis cannot be conveyed to cattle, have proved to be erroneous, for under favourable circumstances human tuberculosis *can* be transmitted to cattle.

Furthermore, as shown by numerous experimenters, various domestic animals, such as goats and pigs, can be infected without diffi-

culty with human tuberculosis, and Herr Schütz himself, working in conjunction with Herr Virchow, obtained positive results, which Virchow described to this Society on the 10th March 1880. To begin with, therefore, we must delete the general term "cattle" from Koch's enunciation; we have really only to deal with the results of experiments on calves.

It is entirely inadmissible to draw such general conclusions as have been drawn from the negative results which Herrs Koch and Schütz obtained in these experiments on calves, for, even before the announcements of the Imperial Hygienic Department, these negative results were contradicted by numerous positive results obtained by experimenters of the most varying kind; and, moreover, the negative could carry but little weight, inasmuch as in this case one positive result obtained under proper precautions is of more significance than a large number of negative ones.

Under these circumstances, it is not the task of the experimenters who have obtained positive results to explain why their results have so fallen out, but of Herrs Koch and Schütz to seek some explanation why they have failed where others have succeeded. They cannot dispose of the difficulty with dogmas like "*si duo facient idem non est idem*" (if two persons do the same thing, it is not the same thing).

I will not at present refer to the earlier positive results obtained by other investigators, but shortly describe the experiments at the Göttingen and Berlin Pathological Institutes to which I have contributed. Last year I described the greater part of the Göttingen experiments in the *Berliner Klinische Wochenschrift*. I exhibited a portion of the preparations at the Congress on tuberculosis. Herr Westenhöffer has already spoken regarding the Berlin experiments in this Society, and later will probably add something new.

The number of these experiments is not large, and the findings are therefore all the more worthy of attention.

Of five calves inoculated directly with tuberculous material which was not obtained from cattle, two developed extensive tuberculosis.

A calf obtained from a presumably healthy byre, and which did not react to tuberculin, died from peritoneal tuberculosis twenty-six days after intraperitoneal inoculation with two fragments of a tuberculous kidney. Not only was the peritoneum covered with tuberculous growth—I show here a piece of the omentum and of the diaphragm—but the mesenteric, portal, and cardiac lymphatic glands, and the axillary, mediastinal, and submaxillary lymph glands, were enlarged, and in part at least could be recognised as tuberculous on naked eye examination. Portions of a mesenteric, a mediastinal, a submaxillary, and a maxillary lymphatic gland were examined, and showed in a quite remarkable way the existence of well-developed tuberculosis with numerous bacilli arranged in layers. I published this important fact in my own journal, and mentioned it at the Congress on tuberculosis, and yet I was forced to read, in a reference of Herr Von Baumgarten's respecting the Congress, that when I had succeeded in producing tuberculosis at all, it had simply been of a localised character, and had shown no progressive character. What then, gentlemen, does one regard as a progressive tuberculosis? If I inject a fluid contain-

ing tubercle bacilli into the abdomen, and a disseminated tuberculosis develops, there can be no question of progressive tuberculosis: but if I insert into the peritoneal cavity two fragments of tissue containing bacilli, and find, not only at one or two spots, but scattered throughout the entire abdominal cavity, tubercles on the peritoneum, that is clearly a progressive change. This, however, was not all; not only the lymphatic glands in direct connection with the abdominal cavity, but those also external to it, including the axillary, mediastinal, and even the submaxillary glands, were tuberculous, containing, as shown by the specimens exhibited, granulation tissue, giant cells, and great numbers of tubercle bacilli. And that is not progressive tuberculosis! That is a disease, according to Von Baumgarten's view, which I might also have produced had I killed the tubercle bacilli in the kidney tissue before using it for experiment!

As Von Baumgarten's abstract on the source of the tubercle bacilli employed in this experiment contains erroneous statements, I may here shortly say that the tubercle bacilli used were obtained from a cavity in the lung of a human phthisical adult, and were purposely not used at once, but only indirectly. They were passed through a guinea-pig, cultivated on a medium of brain substance, and then inoculated into a rabbit, from which was obtained the kidney with which the calf was infected.

My co-worker, Herr Professor Esser, had known for decades the herd, which was certainly subjected to the tuberculin test for the first time by us. The results were negative. Three other calves from the same herd remained free of tuberculosis, although two of them shared the same premises and food as the experimental animal. The feeding was carried out under the continuous control of gentleness from the veterinary school. The changes started from the point of infection. The infective material was prepared with the greatest care, and every precaution was observed to make sure that the bacilli originally derived from the human lung, and no others, were introduced into the calf's body. In short, I am obliged to regard it as proved that in this case bacilli originally derived from a tuberculous human being set up in a calf a progressive tuberculosis.

That such an infection may also be caused by direct inoculation is shown by experiments made in the Berlin Pathological Institute. What, by an unfortunate mischance, could not be attained in my experiment in Göttingen, the proof that the bacilli present in the calf were living and of full virulence, has here been given in a very distinct way. In addition to rabbits and guinea-pigs, I infected two calves which had given no reaction with tuberculin; on being again tested with tuberculin, after a short interval, both reacted. A week ago one of these was killed, and some of you who were present at the former meeting were able to convince yourselves that three-and-a-half-months after intramuscular inoculation there was widely distributed progressive tuberculosis of the lymph glands, and centres in the lungs, in which tubercle bacilli could be detected. The preparations are here again to-day, and you can easily see that the tuberculosis has originated at the point of inoculation, and that the lymph glands in this second calf also, as in the animal directly inoculated, exhibit the same rapid calcification which is so characteristic of perlsucht, that is to say, their condition does not resemble that

common in human tuberculosis, but genuine *perlsucht*, although the infection was with bacilli derived from man.

One might, however, say—in fact it has been said—that these bacilli were originally derived from a child with tuberculosis of the bowel, that they were originally of bovine origin, and therefore that there is small wonder at their attacking calves. Very good; in that case we would have the proof that bacilli derived from cattle can be transmitted to man and can produce well-marked tuberculosis. One may regard the case as one will, but one cannot escape its logical conclusion, viz., that man and cattle may contract progressive tuberculosis by infection with bacilli of one and the same origin.

The Society has been told that the experiments instituted by the Imperial Hygienic Department have also fully confirmed earlier experiences. In spite, however, of the unfavourable method of infection adopted with full intent, four, *i.e.*, 10 per cent., of the varieties of bacilli derived from human sources were found to produce progressive generalised tuberculosis in calves, and not in one calf alone in each series, but in some series in three or four calves in a perfectly regular manner; some of these varieties of bacilli, moreover, when intravenously injected, killed calves by generalised tuberculosis in a few weeks. Two of these varieties were obtained from children with generalised miliary tuberculosis; one of the two was cultivated directly from the lung, so that here also not the shadow of a doubt can exist that the same variety of bacilli which produced generalised tuberculosis in man was also capable of producing a like disease in calves.

I might also draw attention to the fact that, in addition to these four, seven other varieties (*i.e.*, 18 per cent.) of human bacilli, part of which were obtained from adults, proved by no means harmless to calves; on the contrary, in spite of the unfavourable method of inoculation (subcutaneous injection) these proved capable of producing severe tuberculous changes in neighbouring lymph glands, in which still living bacilli were present four months later. Gentlemen, if we declare a child that only has tuberculous changes and tubercle bacilli in the glands of the neck or in the bronchial glands tuberculous, we must certainly say as much of this calf, and we are further justified in describing it as tuberculous, inasmuch as such localised *perlsucht* occurs spontaneously in cattle, and as other experiments—including those of the Imperial Hygienic Institute—show that these varieties of tubercle bacilli when introduced into the bodies of calves by other paths, such as the veins and peritoneal cavity, are probably capable of producing well-marked changes. Of thirty-nine varieties, then, eleven at least, *i.e.*, about 28 per cent., have proved virulent when injected under the skin in calves, whilst, of seven *perlsucht* varieties, one (*i.e.*, 14½ per cent.) proved incapable of setting up severe progressive tuberculosis in these animals. These results, then, together with those obtained by other investigators, show both that bacilli derived from human sources can produce progressive tuberculosis in calves when subcutaneously injected, and also that bacilli obtained from calves may fail to produce the disease when subcutaneously injected into other calves—in fact, that the subcutaneous infection of calves is not a means of certainly distinguishing between bacilli derived from man and those derived from cattle. Further—

more, they enable us to say that human tuberculosis may be conveyed to cattle and cattle tuberculosis to man. The practical questions whether man often contracts tuberculosis from cattle or cattle from man, and how great is the danger to man from tuberculous cattle, are not yet solved, but still call for further careful examination. One thing, however, is certainly clearly demonstrated, viz., that they cannot from the outset be answered in the negative.

EXTRANEOUS SOURCES OF INFECTION IN OUTBREAKS OF ANTHRAX.

By J. M'FADYEAN, Royal Veterinary College, London

DURING the past year more outbreaks of this disease have been reported than in any equal period since it was scheduled under the Contagious Diseases (Animals) Act. In 1902 the outbreaks numbered 687, and the number of animals attacked 1042; with three weeks of this year still to run, the outbreaks since January last amount to 712 and the animals attacked to 1060. It will be observed that the increase, although disquieting, is not alarming. A matter of about 1000 deaths from anthrax among the whole of the domesticated animals in the kingdom, in the course of a year, is, when compared with the losses which some other diseases have inflicted on the flocks and herds of the country, not very serious; and even now there are few countries in which the disease is not more prevalent than it is in Great Britain. It must, however, be admitted that it is unsatisfactory, and in some respects remarkable, that almost ever since the disease was taken in hand, under the Diseases of Animals Act, it has appeared to be increasing. The circumstance certainly warrants careful inquiry as to the probable cause of this increase, in spite of the measures that have been enforced with the object of checking it.

A question that arises at the outset is whether the increase is real or only apparent. Unfortunately there appears to be no reason to doubt that it is real. It has never been possible to accept the official returns as a perfectly accurate representation of the extent to which the disease occurs from year to year, for it is very well known that in the past there has been a good deal of looseness in diagnosis, with the consequence that some cases of anthrax were overlooked, while cases of other diseases were wrongly returned as anthrax. But it is not improbable that the errors in these opposite directions counterbalanced each other, and, at any rate, nothing is known to justify the view that the steady increase in the reported outbreaks in recent years is due to the notifying of cases that were formerly concealed or undetected. The statistics furnished by the Board of Agriculture are probably not accurate now, but there is no reason to think that the margin of error is large, or that it is larger now than formerly.

Accepting the view, therefore, that anthrax is really becoming more prevalent in this country, two possible explanations of this increase suggest themselves. The first is that the increasing number of cases represents what might be called the natural multiplication of the anthrax bacillus within the limits of the United Kingdom. When

once this microbe has obtained a footing in any country, there are probably two different ways in which it may increase and multiply. In the first place, provided the conditions of climate are favourable, it may multiply in the soil, as myriads of perfectly harmless bacteria do almost everywhere. There are, however, two reasons for believing that in this country the multiplication of anthrax bacilli does not go on to any great extent in the soil. The first of these is that throughout at least half the year the temperature is too low. At temperatures below 60° F. the anthrax bacillus is incapable of multiplying, and in order that it may be able to form its spores the temperature must approach 70° F. It is evident that the absence of these necessary conditions for the greater part of the year in temperate climates imposes a serious check on the growth of the anthrax bacillus as a soil organism.

The second reason for believing that the bacillus does not flourish in the soil in this country is that for many years, possibly for many centuries, the country has not been free from anthrax, and that therefore if the conditions had been congenial it would, long ere this, have been so widely distributed in the soil as to make anthrax an exceedingly common disease. In other words, the fact that, after all these years of opportunity on the part of the bacilli, the disease is still not a very common one proves that with us the multiplication of the bacillus as a soil organism does not take place to any great extent.

There remains for consideration the other way in which the bacillus which is the cause of anthrax can multiply, namely, in the bodies of animals affected with the disease. Figures can hardly be employed to convey an idea of the extent to which the bacillus can multiply in this way. A very small number of anthrax germs may suffice to infect an ox, and in an animal dead from the disease the tiniest drop of blood may contain many thousands of bacilli. To say that within the space of forty-eight hours a single bacillus may be multiplied in the body of an animal a thousand million times is to greatly understate what commonly occurs.

It might at first sight appear that this multiplication of the bacilli within the bodies of affected animals furnishes the clue to the increase in the number of outbreaks during recent years, but closer examination of the facts will show that the explanation is not quite satisfactory. In the first place, this method of multiplication had been going on for many years before the disease was scheduled, and before any steps whatever were taken to counteract it by dealing in the proper manner with the carcasses of anthrax animals; and yet the earliest returns of the Board of Agriculture indicate that when the disease was first taken in hand it was less prevalent than it is now. Another fact that makes it still more difficult to accept this explanation is the manner in which the increase is distributed over the country. The enormous multiplication of bacilli which takes place in every fatal case of anthrax greatly increases the chance of future cases of the disease if no steps are taken to destroy the bacilli in the carcase, but the increased danger is strictly local. Neglect of proper precautions on any farm tends to increase the number of cases of anthrax on that farm, but involves no appreciable danger to a neighbouring farm, and certainly none at all to widely distant farms.

But the very point of which an explanation is most needed is the erratic nature of many outbreaks, occurring on farms where what appears to be a reliable history, going back for many years, discloses no previous case of the disease.

If, therefore, the rate at which anthrax is increasing cannot be regarded as the result of the natural multiplication of the bacilli either in the soil or in the bodies of affected animals, the only explanation left to fall back upon is that there must be some outside source of infection. One can imagine at least two possible sources of that kind. The first is artificial manure, into which bones and other animal tissues largely enter, and the second is imported feeding stuffs, such as the various kinds of cattle cake, maize, oats, etc. With respect to the first of these, it may be observed that only those manures in which the animal tissues exist in a comparatively unaltered state, such as undissolved bones and bone meal, can be dangerous in this connection, for the strong chemical agents employed in the manufacture of most of the other forms of artificial manure would probably suffice to destroy any anthrax spores that might happen to be present in the raw materials. But there is still another reason for refusing to regard artificial manures as a fruitful source of anthrax, viz., the erratic way in which outbreaks occur throughout the country, apparently quite unconnected with the more or less liberal employment of artificial manure. For example, in proportion to its cattle population the county of Aberdeen has about fifteen times as many cases of anthrax as Ayrshire, nine times as many as Cheshire, and ten times as many as Shropshire. Similar discrepancies are found on comparing the death rate from anthrax in many of the other counties.

During recent years many veterinary surgeons have acquired the conviction that a good many outbreaks of anthrax in this country must be set down to artificial feeding stuffs (especially linseed and cotton cake) containing the spores of the disease. So far as I am aware, however, no one has taken the trouble to collect and publish observations from which an opinion might be formed as to the justice of the charge laid against such artificial food materials. Obviously the bare fact that feeding cake has formed part of the diet of an animal that has succumbed to anthrax can hardly be said to constitute evidence that the cake in question was the source of infection. On the other hand, by accumulating statistics as to the dietary of animals attacked with anthrax, and other circumstances connected with the outbreak, notably the history or absence of a history of a previous case of the disease on the same field or premises, it might be possible to prove whether any considerable proportion of outbreaks ought really to be laid to the charge of cake and other artificial foods. Circumstances have brought into my possession a certain amount of information of this kind, and I think it well to publish it, although ready to admit that the outbreaks dealt with are too few to justify one in drawing a perfectly positive conclusion with regard to the part played by the use of infected feeding-stuffs in the causation of anthrax among cattle in this country.

The information relates to a series of thirty-nine consecutive outbreaks which have occurred in three English counties in a period of a little over two years. The cases have at least the merit that

there is no doubt as to the accuracy of the diagnosis. In every instance the diagnosis was based on the detection of anthrax bacilli in the blood of an ear or a piece of the tail of the dead animal, one or other of these parts having been sent to me for that purpose, along with a history which is summarised in the following account of each outbreak:—

No. 1.—16th October 1901. A heifer, two-and-a-half-years-old. The animal was apparently quite well on the previous day, but found dead this morning in the field where it was at pasture. It had been bought the previous June, and since that had not had any food but grass. The farm did not comprise any arable land, and there had not been any losses among the cattle on it during the preceding five years. The outbreak came to an end with this death, and there has not been any recurrence of the disease.

No. 2.—2nd November 1901. A cart-horse was found dead in the field on this date. It had been worked on the previous day, and was then noticed to be sluggish. Before being turned out at night, it had been fed with chopped unthrashed oat-straw, but had left part of the feed uneaten. No other case occurred at this time, but in a shippin on this farm a cow had died from anthrax about five weeks previously. The disease has not recurred.

No. 3.—10th November 1901. A five-years-old cow was found dead on this morning. She had given a little less milk than usual on the previous evening, but otherwise had appeared to be all right. The diet was grass, hay, and cotton-cake. No history of any previous case of anthrax on the farm, and the disease has not recurred.

No. 4.—30th November 1901. A two-years-old heifer. First noticed to be ailing this morning at 8 A.M., and died two hours afterwards. The diet consisted of oil-cake, turnips, and hay. Four days later, viz., on the 4th December, a yearling heifer on this farm also died from anthrax. Diet as in the other case. The outbreak then came to an end, and there has not been any recurrence of the disease. There had been three previous outbreaks on this farm, viz., in June 1893, April 1898, and January 1901.

No. 5.—15th December 1901. A three-years-old mare was found dead at 8.45 P.M. She had first been noticed to be ill about an hour previously. Diet, grass, hay, and turnips. There had been several previous cases of anthrax on this farm, the last occurring on 29th October, 1900. No occurrence since December 1901.

No. 6.—20th March 1902. A three-years-old cow. First noticed to be ill about 5.30 A.M. on this date, and died at 4.30 P.M. Prior to death had been fed with bran, hay, and Indian meal. An outbreak of anthrax occurred on this farm about sixteen years previously, and on that occasion a large number of cattle and several horses died. No recurrence since March 1902.

No. 7.—24th March 1902. A cow was found dead in the byre at 6 A.M. The animal had been noticed to be ailing the previous afternoon. Diet, hay, corn, and meal. Six days later a yearling bullock was found dead in a byre at 6 P.M. It had been noted to be ailing during the day. Diet, hay and compound cake. An outbreak of anthrax occurred on this farm in April 1901. No recurrence since March 1902.

No. 8.—30th March 1902. A yearling bullock found dead in a

byre at 6 P.M. Throughout the day it had been noticed to be ailing. It had been fed with hay and compound cake. There was a history of two previous outbreaks of anthrax on this farm, namely, on the 24th March and 7th April 1901. No recurrence since March 1902.

No. 9.—30th March 1902. A two-years-old heifer which at 2 P.M. appeared in its usual health. At 3.45 P.M. she was seen to be ill and was taken into a shed, where she died at 4.30 P.M. The diet was composed of rough pasture, hay, and mangolds. No previous outbreak had occurred on this farm, and no recurrence of the disease since.

No. 10.—1st April 1902. A milch cow was found dead at 6 A.M. Had been seen alive and apparently all right at 10 P.M. on the previous day. At the last milking had given the usual quantity of milk. Diet, linseed cake, mashed oats, bran, mangolds, hay, and straw. No previous outbreak of anthrax had occurred on the farm, and there has been no recurrence of the disease.

No. 11.—6th April, 1902. A cow was found dead in the byre at 6 A.M. It appeared to be quite well at 10 P.M. on the previous day. Diet, oatmeal and cake. No previous outbreak had occurred on the farm. There has been no recurrence of the disease.

No. 12.—16th April 1902. A two-years-old shorthorn heifer, which was turned out into a field with six others at 11 A.M., when it appeared to be quite healthy. At 1 P.M. it was seen at a distance to be lying down, and at 5 P.M. it was found to be dying and expired almost immediately afterwards. Its diet consisted of grass and hay only. The owner stated that he had had a quantity of bone meal lying in sacks in a field for a few days, and that on two occasions, namely, on the 14th and 15th inst., the heifer had been in this field. No history of any previous case of anthrax, and there has not been any recurrence.

No. 13.—28th April 1902. A milch cow which died after about five hours illness. Diet, hay and oilcake. No history of any previous outbreak of anthrax, and no recurrence.

No. 14.—1st May 1902. The stock at this farm comprised seven cows and three horses. The cows stood in two separate shippons, four in one and three in the other. On the morning of the above date one of the lot of three cows was found dead, and by mid-day the cow which stood next to it was also dead. On the following day two of the cows in the other shippon were attacked and died. The fifth cow died a day later, viz., on the 4th May, and the sixth on the 14th of the same month. Thus, only one of the original seven cows survived in this outbreak. The horses remained unaffected. The diet of the cows consisted of hay, ground oats, oilcake, cotton-cake, and Indian meal. There was no history of a previous outbreak, and there has not been any recurrence of the disease.

No. 15.—6th May 1902. A cow was found dead in the byre on this morning. It had appeared to be quite well at 10 P.M. on the previous evening. Diet, grass, hay, and "feeding-cake." No previous outbreak, and no recurrence.

No. 16.—13th May 1902. On this afternoon a cow was found dead. It had eaten its morning meal before it was turned out to grass, but it had refused food on the previous day. In addition to grass, the diet comprised thirds flour, bran, and barley meal. No previous outbreak, and no recurrence.

No. 17.—17th May 1902. The first animal died on this date, after having shown symptoms for about three hours. It was a bull, and had been fed on hay, oilcake, and Indian meal. Nine days later, viz., on the 26th May, a second case occurred, the subject being a cow. It was found dead, but it had been noticed to be ailing a few hours previously. It had had the same diet as the bull, with the addition of barley meal. So far as could be ascertained, this was the first outbreak on the farm, and there has been no recurrence of the disease.

No. 18.—10th June 1902. On the morning of this date a two-years-old heifer was found dead in the field. It had been last seen, on the previous evening, and it then appeared to be quite well. It was being fed with linseed cake in addition to the grass. A previous outbreak had occurred on this farm in July 1899, when one bull died from anthrax. No recurrence since June 1902.

No. 19.—19th June 1902. A cow which had appeared to be quite well on the previous evening died early on this date. It was out at grass at the time. A previous case occurred on this farm in September 1901. No recurrence since June 1902.

No. 20.—30th June 1902. At 2 P.M. on this date a cow was found dead in the field. It had been last seen about an hour and a half previously, and it then appeared to be all right. It had received Indian meal in addition to the grass. No history of a previous outbreak, and no recurrence.

No. 21.—15th July 1902. On this morning a cow was found dead in the field. It had appeared to be quite well the previous evening, when it had given the usual quantity of milk, and been fed with barley meal, rough sharps, and bran. So far as was known, this was the first outbreak on the farm, and the disease has not recurred.

No. 22.—6th August 1902. Early on this morning a cow was found dead in the field. It had given a little less milk than usual on the previous evening, but otherwise appeared to be all right. The diet consisted of Indian meal and "dairy meal," in addition to grass. Believed to be the first outbreak of anthrax on this farm. No recurrence.

No. 23.—3rd September 1902. At 6 A.M. on this date a cow was found dead in the byre. On the previous evening it had been noticed that she did not give her usual quantity of milk, and as it was supposed that she had "caught a chill," a drench was given to her, and she was not turned out to grass with the other cows. The cow had not recently received any artificial food. The present outbreak came to an end with this one case, but a fresh outbreak occurred in November 1903 (see No. 36). In June 1901 a cow was found dead in this same byre, and it was certified to have died from anthrax. The carcase of that animal was buried in the field where the cow which died on the 3rd September 1902 was pasturing.

No. 24.—25th September 1902. On this date a two-years-old bullock was found dead in the field. Along with three others it had been bought at an auction mart on the 20th September, and ever since then it had appeared to be dull. It had not been given any artificial food since it was brought to the farm. No other case of anthrax occurred on the farm at this time, and the disease has not recurred. There was no history of any previous outbreak.

No. 25.—15th October 1902. On this date a four-years-old stallion was unexpectedly found dead in the field where it had been grazing. It had had nothing to eat besides the grass, and no artificial manure had been applied to the field. The present outbreak came to an end with this case. It was ascertained that two days previously, viz., on the 13th October, a two-years-old stallion had been found dead in the same field, and that its carcase had been sold to a knacker. No history of a previous outbreak, and there has been no recurrence of the disease.

No 26.—24th October 1902. Early on this date a cow was noticed to be ill, and about 10 P.M. it died. The cow was at grass during the day, but was also receiving artificial food (nature of the same not stated in the report). No previous outbreak, and no recurrence.

No. 27.—3rd November 1902. On this date a milch cow died, after it had been ill and under treatment by a veterinary surgeon for two days. Since the previous day another cow had been ill, and it was slaughtered on the 3rd.¹ Both cows had been out at grass on fine days, and they were also receiving oilcake, sharps, and hay. It was ascertained that at this same farm a cow had died on the 29th October, after a day's illness, and that its carcase had been sold to a knacker for five shillings, no notice being sent to the police. With this exception, there was no record of any previous outbreak, and the disease has not recurred.

No. 28.—21st November 1902. On this date a cow died after having been ill for about eight hours. The animal had been fed on hay, turnips, oilcake, bran, and Indian meal. Another cow was ailing but recovered. It was ascertained that four days previously a cow had been found dead in the field, and had been buried without notification. With this exception, there was no history of any previous case of the disease, and there has been no recurrence.

No. 29.—24th November 1902. Early on this morning a two-years-old bullock was found dead in the field. It had been only twelve days in the possession of its present owner, and had come from a farm on which there had been several cases of anthrax. Diet, grass and compound cake. This was the first case of anthrax on the field in question, but the field had been dressed with manure taken from premises where several cases of anthrax had previously occurred. There has not been any recurrence of the disease on the above farm.

No. 30.—21st December 1902. A cow died on this date after having been ill for about twelve hours. Diet, Indian meal, bran, and hay. No previous outbreak, and no recurrence.

No. 31.—25th December 1902. On this date a cow was taken ill in the byre, and died within a few hours. The cow had been fed on hay, turnips, bran, sharps, and occasionally a little barley meal. It was ascertained that on the previous day another cow in the same byre had been killed when in a dying state, and when the carcase was taken to the local slaughter-house it was pronounced to be "a case of suspected anthrax." There was no history of a previous outbreak, and there has been no recurrence of the disease.

¹ An ear from each of these cows was forwarded to me for examination. In blood taken from the ear of the one that died anthrax bacilli were detected, but none in the other case. It need hardly be said that this negative result in the case of a slaughtered animal in no way proves that it was not affected with anthrax.

No. 32.—18th January 1903. On this date a cow which had been ill for two days and now appeared to be dying was killed.¹ The diet had been hay, sharps, and Indian meal. Other five cows in the same byre remained healthy. No history of a previous case, and no recurrence.

No. 33.—23rd February 1903. On this date a cow died suddenly. It had been noticed to be slightly ailing for the previous three days. Diet, bran, barley, Indian meal, turnips, and hay. It was ascertained that eight days previously, namely on the 15th February, another cow had been ill, and it had been killed by the farmer and sent to a slaughterhouse, where, on examination of the carcase, it was certified to be a case of anthrax. No previous outbreak on the farm, and no recurrence.

No. 34.—17th March 1903. On the evening of this date a cow died after having been ill for about six hours. The cow had been fed on turnips, hay, straw, corn, linseed cake, and cotton cake. After this cow's death had been reported to the police, it was ascertained that a bull on the same premises had been taken ill on the 13th March, and when he was killed and opened it was noticed that the spleen was ruptured and much enlarged, but the owner stated that he did not suspect anthrax. On the 15th of the same month a heifer died. It was stated that it was ill from the 11th March with a twisted neck, but its death was sudden and unexpected. The animal had also been fed as stated above. On the 17th a pig was unexpectedly found dead in its sty. No *post-mortem* made, and diet not stated. So far as could be ascertained, this was the first outbreak on the farm, and their has been no recurrence of the disease.

No. 35.—10th May 1903. After a few days illness a cow died on this date, and on the following day another cow died. Diet, turnips, hay, sharps, and barley meal. No previous history of anthrax, and no recurrence.

No. 36.—11th November 1903. In the afternoon of this date a cow was found dead in the byre. The cow had been out during the day, and in addition to grass it received oilcake, cotton cake, and sometimes corn and straw. The animal had been noticed to be ill on the forenoon before it died. The outbreak came to an end with this case. There was a history of two previous outbreaks on this farm, namely, one in June 1901, and the other in September 1902 (see No. 23). Only one animal died in each of these outbreaks.

No. 37.—19th November 1903. On this date a cow was found dead in a field. It had been noticed to have been dull that morning before it was turned out to grass. In addition to grass and other materials grown on the farm, the cow had a little cotton cake. No history of any previous case on this farm.

No. 38.—25th November 1903. On this date a cow was noticed to be ill at 8 A.M., and it died about 2 A.M. The animal had not been out of its byre for several weeks, and it was being fed on hay, Indian meal, and feeding cake. No history of any previous cases of anthrax on the farm.

No. 39.—5th December 1903. On this date a cow was noticed to be ill about 8 A.M., and six hours later the owner had the animal killed.

¹ Although this cow did not actually die from anthrax, blood taken from one of its ears showed anthrax bacilli. For comparison see No. 27.

The cow's diet had consisted of hay, Indian meal, and feeding cake. No history of a previous outbreak.

It may now be useful to analyse the principal facts connected with these 39 consecutive outbreaks. Dealing in the first place with the animals that fell victims to the disease, it will be found that these made a total of 54, viz., 49 cattle, 4 horses, and 1 pig. In this connection, the fact that no sheep was attacked in any of the outbreaks is deserving of notice, especially when it is stated that in the three counties over which the outbreaks were distributed there are about three times as many sheep as cattle. The small number of sheep that annually figure in the returns of the Board of Agriculture as having died from anthrax has often been the subject of comment. For instance, these returns show that during the last five years 3390 cattle were reported to have died from anthrax in Great Britain, whereas during the same period only 257 cases of anthrax in sheep were returned. Hitherto it has generally been sought to explain the smallness of the figures for sheep by supposing that a great many cases of anthrax in that species are either undetected or unreported. It appears to be very doubtful whether this explanation is the correct one. One fact that is not in harmony with it is that very few cases of malignant pustule in man are traceable to the skinning and dressing of the carcasses of sheep found dead. Another is that in at least two of the counties in which these 39 outbreaks of anthrax occurred the Local Authorities had taken special steps to call the attention of farmers to the importance of being on their guard against overlooking cases of anthrax in the sheep. Upon the whole it appears to be most probable that anthrax is in reality a rare disease among sheep in this country; and, indeed, there is reason to suspect that the official figures rather overstate than understate the number of cases annually occurring, for hitherto the diagnosis has been based on a microscopic examination of the blood in only a very small minority of cases, and it is well known that in sheep dead of anthrax the alterations observable with the naked eye are much less characteristic than in cattle. It is therefore not unlikely that in a considerable number of cases reported as anthrax in the sheep the disease is of another nature.

It need hardly be said that if in this country very few sheep die from anthrax the fact cannot be ascribed to a marked degree of resistance to infection on the part of animals of that species, for experiments indicate that sheep are considerably more susceptible to anthrax than cattle. One is thus driven to conclude that the comparative immunity of sheep from anthrax in this country must be due to the fact that they are but little exposed to the risks of infection. Like some of the other circumstances hereafter to be mentioned, this appears to point to a special danger attaching to the use of cake and other artificial food-stuffs.

To a large extent what has just been said with regard to sheep applies to horses also. In the thirty-nine outbreaks here dealt with the victims included four horses, but in general the proportion of these animals attacked as compared with cattle is very much smaller. For example, during the last five years only 192 cases of anthrax in the horse have been reported, as against 3390 cases in the ox.

Turning next to the forty-nine cattle included in the above thirty-nine outbreaks, an interesting fact appears when the ages of these

animals are examined. It will be found that these forty-nine cattle comprised thirty-six cows, two bulls, seven heifers and bullocks aged two years or more, one heifer whose age was not specified, and three yearlings. Thus the great majority of the animals were adults, and no animal under one year old was attacked. It need hardly be stated that on many of the farms on which the outbreaks occurred young as well as adult cattle were kept, and the escape of animals under one year old cannot be ascribed to any special resistance to infection offered during youth. It is certain that young animals are as easily infected with anthrax as adults, and it is even possible that there is a greater susceptibility to the disease during early life. Here again it appears to be not improbable that the escape of the younger stock may be due to the fact that they are less frequently than older animals fed with cake and other artificial foods of that kind.

In any attempt to trace the source of infection in an outbreak of anthrax one naturally inquires at the very outset whether there is any history of a previous outbreak on the same field or premises. The information obtainable with regard to that point in these thirty-nine outbreaks showed that in ten of them there was such a history, and in the remaining twenty-nine outbreaks, so far as could be ascertained, there had not previously been any case of anthrax on the farms in question. It would, of course, be rash to accept this absence of any history of a previous outbreak as conclusive evidence that these farms had hitherto been free from anthrax, for some cases of the disease may have been overlooked and others may have been concealed. On the other hand, there is no reason to suppose that the margin of error on this head was very large. In this respect these thirty-nine outbreaks are probably not at all exceptional, for it has long been recognised that outbreaks of anthrax often occur in premises and on farms that have previously been free from the disease.

In the ten cases in which the outbreak was a recurrent one, the simplest explanation would be to suppose that anthrax spores had maintained their vitality on the place in the interval between the two consecutive outbreaks, and this explanation may readily be accepted for a few of the cases. At the same time, it may be pointed out that even consecutive outbreaks on the same farm may quite well be independent, and due to the introduction of fresh virus from some outside source. There is therefore an interest in noticing what was the nature of the diet of the animals attacked even in the ten outbreaks where the outbreak was not the first one. It appears that in four of these outbreaks the animal attacked had been fed only with ordinary farm produce, and in these four cases there was therefore no evidence of a fresh introduction of the disease. In the other six, however, artificial food was also being used, and there is thus a clear possibility that in these the outbreak was independent of the antecedent one on the same farm or premises.

Taking in the next place the twenty-nine outbreaks in which there was no history of a previous case of anthrax, it will be observed that in twenty of these the animal attacked was receiving a diet which included some form of artificial cake or Indian meal. In the other nine outbreaks no such materials had been given to the animals attacked.

Adding together the two series, artificial food was being used in

twenty-six out of the thirty-nine outbreaks. The fact is certainly remarkable, for it is hardly open to doubt that throughout the year only a minority of the cattle in this country receive either cake or Indian meal.

The term "artificial food" as used above covers only cake and Indian meal, but in some of the outbreaks classified as occurring among animals fed with ordinary farm produce only, the diet included such materials as bran and sharps, and it is possible that some of these were of foreign origin. It is obvious that if cake and Indian meal are dangerous in this connection, that is not because of the manufacturing processes to which they have been subjected, but because the original raw materials are to a large extent imported from countries in which anthrax is much more prevalent than it is in Great Britain.

Altogether there were nine outbreaks in which it may be assumed that the diet of the animal attacked was exclusively home-grown, and these were as follows:—

No. 1.—Animal fed on grass only, and no previous outbreak. This case is representative of those very puzzling outbreaks in which no clue to the source of infection can be found.

No. 2.—In this case the diet was composed of grass and unthrashed oats only, that is to say, purely home-grown materials; but five weeks previously a cow had died suddenly in the same byre, and it may be assumed with much probability that this also was a case of anthrax. The first case was therefore probably the cause of the second, and the former may have been due to feeding with infected foreign materials.

No. 5.—Here again the animal attacked had recently received only home-grown produce—grass, hay, and turnips, but there was a history of several previous outbreaks on the farm. It is therefore probable that the infection had not, at least in the later outbreaks, been derived from an extraneous source.

No. 9.—Diet, grass, hay, and mangolds only. The remarks made above with regard to No. 1 apply to this case also.

No. 12.—In this case the animal had been fed with grass and hay only, and there was no history of a previous outbreak. Possibly the bone-meal to which the animal had access may have been the source of infection.

No. 19.—In this case the animal attacked was out at grass, and the report furnished did not expressly state that it had not received any artificial food. Besides, there had been a case less than twelve months previously,

No. 23.—Here the circumstances were almost identical with these mentioned in connection with No. 19. The diet was grass only, but there had been a case of anthrax in the same byre in the previous year, and a third outbreak occurred in the following year.

No. 24.—Although this case has been classed with those in which only farm produce was being given, the animal was only five days in the possession of its last owner, and the history makes it probable that it had been infected before it was sold, possibly by some artificial article of diet.

No. 25.—The two horses which fell victims in this outbreak were at grass, and had not received any artificial food. There was no history

of a previous case of anthrax on the farm, and the outbreak therefore falls into the same category as Nos. 1 and 9.

As has been already admitted, thirty-nine is too small a number of outbreaks from which to draw a very positive conclusion with regard to the alleged danger of artificial feeding-stuffs as a cause of anthrax, but any unbiassed person will probably concede that the circumstances connected with the series of outbreaks dealt with above deepen the suspicion which already existed, that a considerable number of cases of the disease in this country have an extraneous source of infection, and that the vehicle of infection is not infrequently some artificial food-stuff of which the raw material is derived from a foreign country.

THE DETECTION OF TUBERCULOSIS IN COWS BY THE EXAMINATION OF EXPECTORATE.

By JOHN RIDDOCH, M.R.C.V.S., Edinburgh.

TUBERCULOSIS occurs more frequently in dairy cows than in any other class of animal, this being largely due to their close confinement, and the heavy drain on their systems in the production of milk. The tuberculin test has shown that it exists to the extent of from 20 per cent. upwards, the disease being more common in some districts than others.

In the majority of cases the disease is of small extent, and can only be detected by the tuberculin test. There are two conditions, however, in which the diseased animals are a source of danger: *first*, when the udder is invaded and tubercle bacilli occur in the milk, and, *second*, when the lungs are invaded, a condition of more frequent occurrence, and purulent matter containing tubercle bacilli is coughed up and bespatters the wall in front of the animal.

Dried tuberculous sputum blown about in dust is said to be the most frequent means of infection in the human subject, and may not dried purulent matter coughed up by a tuberculous cow and blown about in a byre be a means of infecting the other cows as well as contaminating the milk during milking hours?

When tuberculosis in the udder has advanced to that stage in which an induration can be felt by external palpation, a careful microscopical examination will almost invariably reveal tubercle bacilli in the milk. Indeed, after examining milk from all kinds of abnormal udders for a number of years, I do not remember a case of an indurated tuberculous udder in which by centrifugalizing the milk and carefully examining the deposit tubercle bacilli could not be demonstrated.

It is more difficult to obtain diseased products from the lungs for examination. These products are, however, very frequently available, and afford a means of demonstrating the presence of tubercle even when no other physical symptoms manifest themselves. It is quite true that cows generally swallow their sputum, but if a tuberculous cow is noticed coughing with her head in a horizontal position, her mouth open, and in close proximity to the wall, and if the wall is then carefully scrutinised, small specks of muco-purulent matter will often

be seen adhering to it. These specks of matter contain tubercle bacilli, and afford a certain means of diagnosing the disease, which, so far as I know, has not hitherto received the attention which it deserves.

Perhaps the best way to show the value of this means of examination is to cite a few of the cases which have happened here within the last few months. (See also Fig. 1, Plate IV.)

CASE I.—This was a cow in a byre of forty-eight. She was in rather poorer condition than her neighbours, temperature normal, breathing slightly accelerated. When passing her she was noticed to cough loudly. On proceeding to auscultate the chest two or three patches of muco-purulent matter were noticed on the wall a little above the feeding trough. One of these was scooped up, taken to the laboratory, and microscopically examined. It was found to be very rich in tubercle bacilli. The cow was ordered out of the byre, and slaughtered the following day. The lungs were found to contain a number of cavities (*vomicæ*) filled with the same kind of matter which the animal had coughed up in the stall, and which bespattered the wall in front of her. The mesenteric glands were enlarged and caseous.

Cavities in the lungs of the cow are, however, not of very common occurrence, but this is not the only condition in which tubercular matter is coughed up, as the following cases will show.

CASE II.—This was a cow in a byre of twenty-seven, and she was in fair condition. She had been off her food, but her appetite had returned. She was said to be recovering from stomach staggers. She had a soft low cough, and after coughing her mouth remained open for a short time. The wall in front of her was carefully scrutinised, and after some search a very small speck of matter was found. It contained tubercle bacilli, but not in great numbers. The cow was ordered out, and killed the following day. Both lungs were congested and full of miliary tubercles. The mediastinal and bronchial glands were enlarged and caseous. The mesenteric glands were slightly affected.

CASE III.—This was an animal exposed for sale as a milk cow. She was only a few days calved, was in good condition, and had a fine appearance of milk. The breathing was considerably accelerated. Some small patches of muco-purulent matter were noticed on the wall in front of the animal, and these on examination were found to contain considerable numbers of tubercle bacilli. This cow was sold to a dairyman, and promptly ordered out of the byre next day, when one quarter of the udder was found to be invaded, the milk containing tubercle bacilli. This cow was taken by the original owner to England!

CASE IV.—This animal was one of a herd of thirty-eight, and was in poorer condition than her neighbours. She was noticed to cough, and on searching the front wall some patches of "sputum" were noticed. On examination these were found to be rich in tubercle bacilli. The animal was slaughtered in a neighbouring slaughterhouse the following day, and the carcase condemned for tuberculosis.

CASE V.—This was one in a byre of twenty-eight cows. She was in good condition. She was said to be "taking her food well, and a grand milker." Her breathing was accelerated, and she was noticed to cough. A search on the front wall revealed some specks of muco-purulent matter, and these on examination were found to contain considerable numbers of tubercle bacilli. The animal was ordered out,

and killed two days afterwards in a neighbouring slaughterhouse, and the carcase condemned for tuberculosis.

CASE VI.—This was a cow in a byre of eighty. She was in fair condition. She had a cough, and the owner had blistered her throat with mustard. When examining the animal's throat I noticed small patches of "expectorate" on the wall. One of these was scooped up and examined. It contained large numbers of tubercle bacilli. This cow was ordered out, and her condition described to the owner, who sent her to the knackery the following day.

CASE VII.—This was a cow in a byre of seven. She had been off her feed, and was put into a stall by herself. Some mucopurulent matter was noticed on the animal's back which she had coughed up in the act of licking herself. This matter was very rich in tubercle bacilli. The cow was ordered out, and slaughtered the following day. Both lungs were full of miliary tubercles; they also contained softened caseous matter in several places. The mesenteric glands were enlarged and caseous. One quarter of the udder was invaded, but the teat had been amputated and no milk could be obtained from that quarter.

CASE VIII.—This was a cow seized in the fat stock sale. She coughed occasionally with a loud strong cough. She was in good condition and had a glossy coat. Some specks of matter were found on the wall, and these contained tubercle bacilli. The owner of this cow was so confident that she was free from disease that he got two veterinary surgeons to attend at her slaughter on his behalf. The pleura and peritoneum were covered with granular tubercle, and the lungs contained softened caseous matter. The mediastinal glands were enlarged and caseous. The mesenteric glands were also invaded.

CASE IX.—This was a cow in a byre of ten. There was slight heaving of the flanks, and her temperature was 104° F. One small patch of expectorate was noticed on the wall. On examination this was found to contain tubercle bacilli. The owner said he had never seen anything amiss with the cow, and that she was milking well. She was immediately ordered out, and was slaughtered two days afterwards. The lungs of this animal were badly affected, and the mesenteric glands were enlarged and caseous. The pleura, peritoneum, and other organs were entirely free.

CASE X.—This was a cow in a byre of twenty. The owner drew my attention to this animal, as she had improved so much since my last visit. On approaching the cow to examine her more closely, I noticed two small spots of expectorate on the wall, and these on examination showed tubercle bacilli. The cow did not cough during the time I was in the byre. The owner of this animal refused to believe that there was anything amiss with her. She was ordered out, and slaughtered the following day. The lungs contained a number of caseous centres, and a mass of tubercular deposit about eight inches in diameter was adhering to the right costal pleura.

CASE XI.—This cow was ravenously eating turnips during my visit, and on passing behind her I noticed her cough; this led to an examination of the front wall. Several patches of matter were observed, and these were found to contain large numbers of tubercle bacilli. This cow was ordered out, and finally sent to England.

CASE XII.—This was a cow in a byre of sixteen. Her breathing was accelerated, and after watching her for a short time she was noticed to cough. An examination of the front wall revealed some specks of matter, and these on examination showed tubercle bacilli. The owner of this animal was indignant when he was told that the cow was tuberculous. He maintained that she was in perfect health and a heavy milker. She was ordered out, and was slaughtered two days afterwards. The lungs contained a quantity of softened caseous matter throughout their substance. The mediastinal and bronchial glands were enlarged and caseous; the mesenteric glands were about the size of hens' eggs and contained caseous matter. The other organs were not invaded.

In the above cases the expectorate was carefully scooped up in a piece of paper torn from a note-book; the paper was then rolled into a scroll and placed in a sterilised test tube, the test tube being closely corked to prevent desiccation during transit to the laboratory.

More cases might be cited, but these will suffice, the object of this paper being to show the value of this method of examination.

A cow will occasionally cough up purulent matter in other conditions than tubercle, such as a common catarrh, but an examination of the expectorate in these cases will show only catarrhal cells and the complete absence of tubercle bacilli.

[I have had an opportunity to examine Mr Riddoch's preparations, and can vouch for the accuracy of his observations. It may be well to add that there is here no question of "grass bacilli," as the preparations show that the bacilli always occur together with well-staining leucocytes and other cells derived from the lesions or from the air-passages.—J. M'F.]

A FURTHER NOTE WITH REGARD TO THE STAINING REACTION OF ANTHRAX BLOOD WITH METHYLENE-BLUE.

By J. M'FADYEAN, Royal Veterinary College, London.

IN this *Journal* for March last (p. 35) I described the peculiar colour reaction which is obtained when an imperfectly fixed film of blood containing anthrax bacilli is stained with aqueous solution of methylene-blue. The fact that such a peculiar and striking reaction was obtainable in this way with anthrax blood had been known to me for a number of years, and during the whole of that time I had never failed to obtain it with more or less distinctness according to the number of bacilli present. It was therefore with some surprise that on a recent occasion I failed to obtain the reaction described with a preparation of anthrax blood in which the bacilli were abundantly present. As this was the first preparation stained with a freshly-made 1 per cent. solution of Grüber's methylene-blue, it was immediately surmised that the uniform success which had formerly attended the use of the stain must have been due to some impurity in the dye used. This appeared to be the more probable since for a number of years past the solution in use in the Laboratory had been from time to time made from the same stock-bottle of methylene-blue powder.

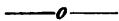
This stock, indeed, had been in use for several years, and I am unable to say from whom it was originally obtained. The failure to obtain the reaction with the freshly-prepared solution of presumably pure methylene-blue was a little disconcerting, but, fortunately, the explanation of the discrepant results was soon discovered. A 1 per cent. solution of fresh Grüber's powder was prepared by boiling, with the addition of a $\frac{1}{2}$ per cent. bicarbonate of soda, and this was found to give the specific reaction with great brilliance. Apparently therefore the stock of methylene-blue which had been in use in the Laboratory for a number of years either had the polychromatic quality originally owing to some impurity, or it had acquired it by keeping. Subsequently it was found that a freshly purchased sample of Merck's pure medicinal methylene-blue gave the reaction without the addition of bicarbonate of soda.

With this explanation the directions given in the previous note on the subject may be allowed to stand. Further experience only serves to impress me more with the practical value of this method of staining in the diagnosis of anthrax. Alike in respect of simplicity, and in the characteristic appearance of the picture which it produces in the blood film, it is much superior to any other method yet devised, not omitting those directed towards the staining of the capsules of the bacilli. As previously stated, the reaction is macroscopic as well as microscopic, and this to such an extent that in dealing with ordinary samples of anthrax blood from the ox, sheep, or horse, in which the bacilli are nearly always very numerous, one can generally recognise the peculiar reaction with the naked eye. Conversely, when the sample of blood being examined comes from one of the animals mentioned, and it is not putrid, one can with very little chance of error pronounce the case to be not anthrax if the stained film lacks a purple shimmer, and presents only a greenish-blue tint. It need hardly be said that these remarks must not be construed as a recommendation to dispense with microscopic examination of the stained preparation; they are merely intended to emphasise the remarkably distinct nature of the reaction.

As the reddish-purple reaction is due to the staining of the disintegrated capsules of the bacilli, it is entirely prevented by any procedure which fixes the capsules, and care must therefore be taken not to heat the film too strongly. For the same reason, the distinctness of the reaction depends upon the richness of the blood in anthrax bacilli, and in cases of that disease in which a general invasion of the blood stream had not taken place at the time of death this method of staining is obviously of no value. There is, however, probably only one domestic animal in which death may occur from anthrax before this general invasion of the blood has taken place, viz., the pig. In an animal of that species failure to detect anthrax bacilli in the blood by any method of staining does not warrant one in concluding that the case is not one of anthrax. In recent cases of anthrax in the pig, however, I have found the methylene-blue method of staining very valuable when applied to scrapings taken from the lymphatic glands of the throat.

In conclusion, it may be mentioned as a point of interest, though not of importance, that the reddish-purple tint of anthrax films stained in this way is apt to fade, even in preparations that are protected from light.

EDITORIAL ARTICLE.



WHAT IS THE BEST METHOD OF DISPOSING OF THE CARCASES OF ANIMALS DEAD OF ANTHRAX?

IN an article which appears in the preceding pages of this number some evidence bearing on the cause of anthrax outbreaks in this country is set forth, and it is there shown that in a considerable proportion of such outbreaks the circumstances appear to point to an extraneous source of infection. Otherwise stated, it would appear that notwithstanding a long period of past freedom from the disease anthrax may break out on almost any farm in the kingdom, and, furthermore, that the disease may recur after a longer or shorter interval in spite of attempts made to avert this by the proper disposal of the dead animals and thorough disinfection of the premises in which the deaths occurred. To admit this view of the facts, however, in no way detracts from the prime importance of putting into practice every reasonable measure that is likely to exterminate the seeds of future mischief which are brought into existence locally whenever an outbreak of anthrax occurs.

Impressed with the importance of such measures, the Board of Agriculture have recently issued to Local Authorities in Great Britain a circular in which attention is called to the continued prevalence of anthrax, and some valuable advice is offered with regard to the proper means of prevention. Naturally, this advice extends to what is considered the most effectual manner of dealing with the carcases of animals dead of anthrax. The advice offered by the Board on that head is that Local Authorities should make provision for the destruction of anthrax carcases by the process of burning, or by exposure to a high temperature, "provided this can be done without cutting the carcase." It is suggested that in urban districts this may be done in a destructor, or in a digester; and, to meet the case of country districts, a method is described whereby the carcase may be consumed by fire on the spot where the animal is found dead. In connection with this plan, it is rightly pointed out if the carcase has to be moved this ought not to be done by dragging it along the ground, that the vehicle used for the transport of the carcase should afterwards be disinfected, and that the natural orifices of the body should be plugged to prevent the escape of blood or other infective matter.

From the theoretical standpoint the whole of this advice is excellent, but it may be permissible to suggest that the practical

difficulties in the way of cremating all anthrax carcases are very considerable, and that the superiority of the method over the one more usually adopted is not great, and, as applied to many cases, non-existent. The difficulties which attend cremation are obvious. Where a proper destructor is not available, the Board suggest that the carcase should be consumed by an extemporised fire made with one ton of wood and fifteen cwt. of coal, or of coal and coke, with perhaps the addition of paraffin to assist combustion. The first objection that may be raised to this plan is that it will generally involve considerable delay, which is always undesirable; and the second is that it is much more expensive than the method of prompt burial. These objections apply with even greater force to the suggestion that Local Authorities should make provision for the destruction of anthrax carcases in a destructor or digester. One wonders whether the Board of Agriculture seriously intended that an apparatus of this kind, capable of taking in the intact carcase of a large ox, should be erected in every parish in the country. It is difficult to believe that anything so impracticable can have been recommended, and yet it almost appears as if the erection of multiple destructors in every county was what the Board really intended to advise, for otherwise in almost every case of anthrax it would be necessary to transport the carcase for a long distance, with highly undesirable risks which no precautions can entirely avert. It is, however, unnecessary to dwell upon the manifest disadvantages of the method of cremation in respect of expense and difficulty of execution. The important question is, putting aside the cost and inconvenience, is the method of cremation superior to any other plan of disposing of anthrax carcases? It is by no means certain that this question ought to have an affirmative answer, although it is virtually answered in that sense when the Board state that, "the most effectual manner of disposing of the carcase of an animal dead of, or suspected of, anthrax, is by the process of burning, or its destruction at a high temperature, provided this can be done without cutting the carcase." To advise that an anthrax carcase should be cremated rather than buried, or to say that the former method of dealing with the dead animal is the most effectual, implies that disposal by burial is ineffectual for the purpose in view, which, of course, is to render the carcase non-infective. Apparently the Board of Agriculture believe that the practice of burying the carcases of animals dead of anthrax is not a safe or effectual one. Many people who have given a good deal of thought to the subject believe the contrary. It is very well known that even a moderate degree of putrefaction of a carcase destroys any anthrax bacilli present in it. It is equally true that even advanced putrefaction of animal tissues containing anthrax spores may leave such materials still dangerous, but one may admit that fact without being obliged to prefer cremation

as a general method of disposing of anthrax carcasses. Burial would have to be condemned if there were any reason to think that this plan would in any considerable number of cases involve the deposition of anthrax spores in the soil. But it is a matter of certainty that, provided the burial is promptly carried out, the act involves nothing of the kind, and there is no evidence to show that in temperate climates this method of attempting to avert subsequent mischief is any less effectual than cremation. In short, we believe that the proposal to adopt destruction by fire or a high temperature as the general method of dealing with anthrax carcasses in this country is impracticable, and it is unnecessary, because prompt burial of the intact carcase is comparatively easy to carry out in most places, and quite effectual as a means of ensuring the destruction of the anthrax bacilli present in the body of the dead animal.

We hear that some Local Authorities have been a good deal perturbed because they found it well-nigh impossible to give effect to the suggestions with regard to cremation contained in the circular sent to them by the Board of Agriculture, and because they inferred that to continue to sanction burial as a means of disposing of anthrax carcasses would be to sow the seeds of future outbreaks. There is no occasion for any such anxiety. It is quite certain that if anthrax outbreaks are increasing the cause is not to be sought in the burial of animals dead of the disease, but in sources of infection that remain above ground.

Reviews.

Bacteriology of Milk. By Harold Swithinbank, of the Bacteriological Research Laboratory, Denham, and George Newman, M.D., F.R.S.E., D.P.H., Medical Officer of Health of the Metropolitan Borough of Finsbury, and formerly Demonstrator of Bacteriology in King's College, London. London: John Murray, 1903. Price 25s.

THE conjoint authors of this work are to be congratulated on having conceived and carried into effect the idea of collecting into one volume all the scattered information with regard to the bacteriology of milk. The experience and researches of recent years have brought home even to the lay public the great importance of milk as a vehicle in the dissemination of human diseases, and "milk epidemics" constitute common ground on which medical men and veterinary surgeons are daily brought into contact. For these reasons it is obvious that anyone capable of producing a good textbook on the bacteriology of milk could count beforehand on obtaining a large circle of readers.

The book now under notice is certainly not of the so-called "popular" type. This will immediately be apparent when we say that it is a ponderous

volume, extending to nearly 600 pages. To those who have no previous acquaintance with the subject it may appear strange that anyone, without being guilty of redundancy or repetition, could find matter relating to the bacteriology of milk to fill a volume of this size. Nevertheless it may safely be said that the work which Mr Swithinbank and Dr Newman have produced owes nothing of its size to either redundancy or repetition. That, however, is not to say that it might not with advantage have been made smaller, by omitting to deal with some points which, although they fall quite naturally under the title selected for the work, might perhaps have been left out without exposing it to a charge of incompleteness.

The opening chapter is devoted to some general conditions affecting bacteria in milk, such as its general properties and composition, the physiology of lactation in the cow, fodder and its effect on milk, etc. Chapters II. and III. deal with the technique of bacteriological examination of milk, and are followed by a chapter in which the examination of air and water in relation to milk supply is described. The matter of these three chapters is excellent, but it is questionable whether it would not have been better to deal more briefly with these subjects, which are, of course, discussed in all the text-books on bacteriology.

The succeeding three chapters deal with the bacteriology of milk from what may be called the economic standpoint. The remainder of the book, amounting to two-thirds of the whole, is devoted to the pathological aspects of milk bacteriology, and this is at once the most original and the most valuable part of the work. The exigencies of space on the present occasion will not permit us to outline, or even to mention, the whole of the important subjects that are discussed in these final chapters, but we may say that nothing connected with milk as a factor in the causation of disease has escaped notice. Tuberculous milk naturally comes in for a large share of attention, as do also milk-borne epidemics of scarlatina, diphtheria, sore-throat, and typhoid. The control of the milk supply by the State and by private enterprise are also dealt with at considerable length. Lastly, we must not omit to mention that the book is illustrated by 33 plates and 35 smaller figures in the text.

The work is certain to have a large sale among medical officers of health, and no veterinary surgeon who has to do with the inspection of milk or dairy cows can afford to be without it.

Surgical and Obstetrical Operations for Veterinary Students and Practitioners.

By W. L. Williams, Professor in the State Veterinary College, New York, U.S.A. Pp. 110. Illustrated with thirty-four plates and fourteen figures. Published by the author.

This small work appears to be a revised and somewhat amplified version of Pfeiffer's (University of Giessen) *Operationskursus*, as translated by Professor Williams, and published about three years ago. In many ways the second edition is better than the first. The new book has been enlarged, and its illustrations have been increased and improved. Additional matter is represented by ovariectomy in the cow, bitch, and cat, by nine embryotomy operations, and by a new (*vide* preface) operation for polleveil—of which the remark may be permitted that careful scrutiny of the details gives no clue to newness. Arytenectomy now replaces Merillat's operation for roaring—"arytenoididraphy"—unattractive in name and apparently of small value in practice. Staphylotomy, as an exploratory operation in pharyngeal disease, has been retained, though its performance in the horse is seldom or never justifiable.

Some confusion still continues regarding the relationships of the facial sinuses and the treatment of impaction of these cavities. The trephine opening into the frontal sinus, as shown in Plate IV., is directly over the ethmoid lateral mass and needlessly near the cranial cavity. The position of the partition which usually separates the upper and lower maxillary sinuses is said to be of "scant interest," and probably for this reason the best point for opening the larger maxillary sinus to ensure efficient drainage is not clearly indicated. The introduction states that surgical operations "acquire value only when properly correlated to disease and skilfully performed," but notwithstanding this caution it would seem that in trephining the sinuses the position and number of the holes—nasal or facial—is immaterial. With these and a few other exceptions, the operations, fifty-three in number, are well described; and, while everyone may not agree with the author's explanations of doubtful points, the directions given should afford considerable assistance to the student or practitioner in operative surgery.—J. M'Q.

A Text-Book of Veterinary Medicine. Vol. V. By James Law, F.R.C.V.S. Ithaca. Published by the author, 1903.

THIS is the concluding volume of Professor Law's encyclopædic work on veterinary medicine, of which we have already had occasion to speak in terms of praise in noticing the appearance of the earlier volumes. It is mainly devoted to the animal parasites, but also deals with ringworm, actinomycosis, and other diseases caused by parasitic fungi. It need only be said that the high level of fulness and accuracy which characterised the preceding volumes is maintained in this one, and we congratulate the author on the completion of a work which is a really valuable addition to veterinary literature.

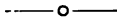
Protozoa and Disease. By J. Jackson Clarke, M.B., London. Part I. London: Bailliere, Tindall & Cox, 1903.

THE modest estimate of his own work which the author of this book expresses in the preface is to some extent calculated to disarm criticism, for it is there stated that to biologists much of the matter must appear rough and elementary, though the hope is expressed that the work may serve to give them an insight into the view of those who are concerned with pathology, and at the same time be useful to medical men "as a basis for considering recent and forthcoming work on the protozoa in disease." Presumably for the benefit of biologists and medical men, the author proposes in a second part to collect the work that has been done with regard to the part alleged, but not as yet fully proved, to be played by protozoa in disease. Candour compels us to say that we form, if anything, a lower estimate of the practical usefulness of the book than the one held by the author himself. "Rough and elementary" are the terms that correctly describe the quality of the matter. It is not to be supposed that any biologist would have the patience to read the author's account of the protozoa here dealt with, and it is likely to prove equally disappointing to the practitioner, whether medical or veterinary, who consults its pages for information with regard to the rôle played by the protozoa in the causation of disease. The book, in short, is on the plane of the "popular lecture," and it cannot be recommended except to those who do not desire more information on any subject than can be agreeably imparted under that guise.

Präparirübungen am Pferd. Von Dr. med. vet. Reinold Schmaltz, Professor der Anatomie an der Thierärztlichen Hochschule zu Berlin. Theil III. Berlin: Richard Schoetz, 1903.

THIS, the concluding part of Professor Schmaltz's dissection manual on the horse, deals with the organs of the thorax, abdomen, and pelvis, and also with the brain, eye, and larynx. To describe the work as a dissection manual is, however, likely to convey, at least to English readers, an inaccurate idea of its plan and scope, for in the arrangement of the matter the book differs considerably from any of the guides to dissection provided for the use of medical or veterinary students in this country. It differs from these mainly in that more space is devoted to the operations necessary for the proper exposure of the various organs, whether for the purpose of ordinary dissection or for *post-mortem* examination in the case of disease, and some objection might be taken to the arrangement of the matter, inasmuch as in most cases detailed descriptions of the anatomy and connections of the organs precede the directions with regard to the operations necessary to bring the different objects into proper view. In all other respects the work is deserving of the highest commendation. It extends to 335 pages, and the illustrations in the text, twenty-four in number, are for the most part excellent. Of even higher merit are four plates, with eight figures, illustrating the anatomy of the brain.

CLINICAL ARTICLES.



POISONING OF CATTLE BY COMMON CELANDINE.

By H. CAULTON REEKS, F.R.C.V.S., Spalding.

ALTHOUGH I have some dim recollection of reading of previous cases of this kind, I find myself unable to turn up recorded instances. It seems that such are few, in which case the history of the following should prove of interest. To commence with, a description of the plant may not be out of place.

Bentham and Hooker, in their fifth edition of *British Flora*, describe it thus:—

"*Chelidonium majus*. *Common Celandine*. A genus of Papaveraceæ reduced now to a single species. Rootstock perennial. Stems erect, slender, branching, one to two feet high, full of a yellow, fœtid juice, and generally bearing a few spreading hairs. Leaves thin, glaucous underneath, once or twice pinnate, the segments ovate, coarsely toothed or lobed, the stalks often dilated into a kind of false stipules. Flowers small and yellow, three to six together, in a loose umbel, on a long peduncle. Pod nearly cylindrical, glabrous, one and a half to two inches long.

"On roadsides and waste places throughout Europe and Russian Asia, except the extreme north. In Britain, chiefly near houses. Frequent in England and in some parts of Ireland, less so in Scotland. Flowers all summer. Common or countryside names for the plant

are many ; and it is known variously as Swallow-wort, Teter-wort, Wart-wort or Wart-weed."

All this contains no mention of its poisonous nature. Other authorities, however, agree in recognising its dangerous properties. For example:—

Woodman and Tidy, *Forensic Medicine and Toxicology*, state that it is poisonous, but do not quote cases.

Bentley, *Manual of Botany*, states: "The Celandine is a native of this country, growing in the neighbourhood of villages. It has an orange-coloured juice of a poisonous nature, which is a popular



Chelidonium majus, with fully expanded flower, buds, leaves, and fruit.
One of the pods dehiscent.¹

external application for the cure of warts, and has been used successfully in opacities of the cornea. It has been also administered internally, and is reputed aperient, diuretic, and stimulant."

Cassell & Co's *Encyclopædic Dictionary* contains: "It is full of a yellow juice which is of an acrid poisonous nature, and has been used in certain diseases of the eye, and as a caustic to destroy warts, etc. . . . Its juice is a virulent acrid poison."

Year Book of Pharmacy, 1897: "Chelidonine is recommended in the place of opiates for the relief of pain in the stomach and bowels, and as a sedative in ulcerative conditions of the stomach."

¹ This illustration is copied from *School Botany*, by John Lindley, M.D., Ph.D., F.R.S.

Other authorities mention among its many alkaloids (some eighteen in number) the presence of the narcotics Chelidonine and Chelerythrine, and the existence of various bitter irritants. It is reputed also to act as a drastic purgative, and to produce in animals eating it vomiting, loss of sight and hearing, inability to stand, and death.

I have thought it well to refer to these in full, as it will be seen from them that the plant contains both narcotics and irritants. With a knowledge of these facts, the symptoms in a suspected case of poisoning from it may be set at their true value, and the absence of purging in the cases I am about to relate accounted for.

History.—3rd October. I received a wire from a client five miles from home requesting my immediate attendance. On my arrival I found two valuable pedigree cows lying dead in the yard, and the remainder of the herd, nineteen in number, perfectly healthy. For some time past these cows had been allowed to graze in a large field adjoining the house, but at night had regularly been yarded for feeding and shelter. The illnesses had occurred while at grass, and had given but little premonitory warning of death.

Knowing anthrax to have been declared on the farm but a short time before, I was naturally suspicious, and declined to hold a *post-mortem* before making a microscopic examination of the blood. So far as anthrax was concerned, my investigation was negative, and I wired the owner to that effect the same evening, after which I received from him no further instructions.

6th October. I met the owner in Spalding, and was surprised to hear that, in order to avoid the spilling of blood on his farm, he had commissioned a veterinary surgeon in a neighbouring town to make the *post-mortem* on these two cows in a knacker's yard. This gentleman's report was unproductive of help, save that he mentioned a slight condition of inflamed fourth stomach.

I now suggested to the owner the advisability of carefully investigating the field in which these animals were grazing, expressing the opinion that it was there the mischief would be found, for a mass of eliminative evidence had already placed the food in the yard beyond suspicion. Our greatest piece of evidence of this description was the fact that a herd in an adjoining yard, all the members of which had kept well, was being identically fed on the same food, with the sole exception of being shut off from this particular field.

My suggestion was treated lightly. The owner was one of those unfortunate individuals whose strength of will far exceeds the supply of ordinary knowledge at their command necessary to guide it into right channels. In other words, he was obstinate. He persisted in putting the outbreak down as "some undiscovered disease" that veterinary surgeons were as yet quite unaware of, and somewhat pooh-poohed, if not altogether ridiculed, the idea of anything poisonous existing in the field which he and his had known for generations. Already I had reluctantly decided to put the cases in my category of "deaths from undiscovered causes." The owner, however, was fated to be compelled to further assist in the elucidation of the mystery. He was called to his senses by the death of two more of the herd.

10th October. Again I was wired for. One cow was dead, another

ill. The herd was again in the yard, having been shut in since six o'clock the previous evening. The ailing cow simply appeared drowsy the remainder of the animals, as before, healthy. Before making the autopsy I decided first to examine the field, and a very short search served to discover large quantities of the celandine in one of the hedge-rows. I immediately pointed out to the owner that this was extremely poisonous, but did not rest satisfied until I had searched the rest of the ground. Finding nothing further, we were thrown back on the celandine as the cause of the mischief, especially as it bore plentiful evidence of being "topped" by the cattle.

It was now that I made the *post-mortem*, hoping to find in the rumen confirmatory evidence in the shape of undigested leaves, stems, or fruits. I was disappointed. Everything was in a pulpy, finely divided condition. I was able, however, to point out to the owner two facts—the very succulent nature of the plant we suspected, and the twelve hours of digestion that had gone on since the animals were shut in from the field. Those facts taken together did not promise the finding of undigested material. As a matter of fact, the only substances that had at all withstood the actions of the stomach juices were the stalks of some dried lucerne and clover, together with the chopped straw the animals had received overnight. This led me now to examine the ailing cow more minutely.

Symptoms.—There was a previous history of a large flow of saliva and inordinate thirst. Now, however, one had great difficulty in picking her out from the rest of the herd. Beyond a somewhat sleepy expression of the eyes and half-closed lids, nothing was to be noticed. When moved, however, the gait was uncertain, and, if pushed, staggering. The bowels were torpid, but the kidneys unusually active, the shepherd remarking that there were three or four of the animals constantly staling, this one among them.

Remembering that the suspected plant was a species of the poppy family, and, fortunately for myself, being then ignorant of its sometimes purgative action, I assumed its poisonous principle to be narcotic, thus confirming to myself the opinion I had already formulated that the celandine was responsible for the illnesses. Thereupon I decided to administer a combined dose of linseed and castor oils, with the addition of *nux vomica* to counteract the sedative action of the poison. This prepared, we attempted to secure the cow. The excitement this caused her, however, appeared to increase the symptoms of intoxication. At the first movement to touch her, with a preliminary frantic bellow, she fell to the ground in convulsions, limbs outstretched and quivering, constant moaning and beating about of the head, and eyes drawn back out of sight into the orbits. From this she appeared somewhat to rally, at the end of ten or fifteen minutes endeavouring to sit upon her sternum, and looking round with signs of returning consciousness. Even now a near approach to her served to bring about a repetition of the convulsive state. I ventured, however, upon a hypodermic dose of a stimulant, unhappily without effect. An hour or two later she was dead.

I should mention in passing that each of these cows was suckling a calf, and that in neither instance was the calf affected.

Treatment.—The remaining eighteen cows were treated with saline and oleaginous purgatives.

11th October. I visited the herd again, found the purgatives operating upon some, but not all, and, with the exception of slight symptoms of sleepiness in one or two, the other cattle healthy.

12th October. One of the cows was still exhibiting these symptoms of drowsiness, and had not yet responded to the purgative. A further dose of castor oil was given her.

13th October.—This cow was still ill. From now until 16th October she gradually grew worse, showing marked symptoms of gastro-enteritis and bladder or kidney irritation. On the latter date an offensive purge set in, and later in the day she expired from exhaustion.

Remarks. It would appear from what I could gather from the men on the farm that these cows had been in the habit of picking at this plant more or less all the summer, and without ill effects. From this it is fair to assume that in all probability the poisonous principles within it reach their height of activity in the autumn, that is to say, when the plant is in full fruit. With many of our poisonous plants I think that is a point to be remembered.

Another point of interest to veterinary surgeons is this—had this been a herd of cattle other than cows these cases would possibly not have happened. Cows are proverbially mischievous, and for what reason they should forsake the ten to twenty acres of luxuriant herbage for the poisonous growths of a hedgerow is a point only to be appreciated by those who are well acquainted with their habits.

These cases also form a striking illustration of the manner in which a number of small causes combine to a great one. The hedge—some 120 yards of it—in which the celandine was growing had in the early part of the summer been what is called in this district “split.” It had been divided down its centre, and one half of it, that on the inside of the field, removed. This operation had given to the celandine the benefit of a more than ordinary supply of fresh air, moisture, and sunlight. The result was a luxuriant and extraordinarily robust crop. A further point still, it had been placed still more within the reach of the cattle in the field.

A CASE OF “BUTTRESS FOOT,” OR FRACTURE OF THE PYRAMIDAL PROCESS OF THE PEDAL BONE.

By A. R. ROUTLEDGE, M.R.C.V.S., London.

THE subject of this note was a chestnut mare, nine years old, and used for omnibus work.

History.—For about two months the mare was lame on the off fore leg, and in spite of treatment the condition became steadily worse. The off fore foot was rather long and narrow, and the fetlock joint was inclined to be bowed outwards, but the degree of lameness was out of proportion to these defects, and the diagnosis was obscure.

Median neurectomy was performed on the 10th May 1902, and reduced the lameness to about half of what it was before. On the 5th June ulnar neurectomy was performed, with the result that the mare became sound and went to work three weeks later. She continued to work soundly and well, being inspected from time to time.

During February of this year the coronet began to enlarge in front and slightly to the outer side, and gradually a ridge of bone grew down from the coronet to the toe. The case, in fact, became a typical one of so-called "buttress foot," which my friend Mr. Willis has described as diagnostic of disease of the pyramidal process of the pedal bone. Meanwhile the swelling of the coronet, which appeared to be mainly composed of fibrous tissue, increased in size, until the whole of the front and sides became involved, assuming the appearance shown in Fig. 1.



FIG. 1.

In spite of the coronary enlargement, the mare worked well, and remained free from lameness till the 8th June last, on which day the limb became swollen up to the site of the median operation. The appearance of the limb closely simulated an attack of lymphangitis. The mare was kept under observation till the 13th of the same month, during which time the swelling increased, as did also the lameness to a slight degree. During progression she brought the heel to the ground and "rocked the toe" as in a case of rupture of the perforans tendon. The mare was killed on the 13th June.

Post-mortem.—In trying to pull away the hoof from the sensitive structures with a pair of farrier's pincers, the tendons and ligaments of the corono-pedal articulation gave way, leaving the pedal bone *in situ*. The flexor perforans tendon showed inflammatory softening, and was very nearly ruptured through at the level of the navicular bone. There was slight evidence of navicular disease. The articular cartilage of the corono-pedal joint had been almost completely removed, and there was sclerosis of the opposed bony surfaces,



FIG. 2.

which, by unequal wear, had brought about deformity of the os coronæ and os pedis.

There was very old-standing fracture of the pyramidal process (see Fig. 2), with the formation of a false joint between the process and the pedal bone. There was also a recent fracture of the part of the pedal bone which carries the articulation for the navicular bone, and this and the tendon lesions probably accounted for the final symptoms of "break down."

Neurectomy enabled us to get a year's useful work out of what would otherwise have been a hopeless cripple.

AN OUTBREAK OF VENEREAL DISEASE AMONG SHEEP.

By W. H. FLOOK, M.R.C.V.S., Rickmansworth.

THE following is the history of a somewhat remarkable outbreak of disease among sheep which recently came under my observation.

On the 15th August last fifty-two yearling ewes and two ram lambs were purchased and brought to a farm in this locality. Some after their arrival it was noticed that there was a discharge from the sheath of one of the ram lambs, and that the other had a rather



extensive eruption around the mouth and nose. Although it was not known for certain the shepherd believed that these lambs had been serving the ewes for a week previous to their arrival. The two rams were separated from the fifty-two yearling ewes and put with a small flock of older ewes. A few days later it was noticed that a disease affecting the vulva had attacked a number of these old ewes, and the rams were therefore taken away from them. They had been with the ewes altogether about a week.

All this had happened before I was first called in, which was on the 30th August. At this date I found that altogether nine of the old ewes were affected. There was great swelling of the vulva, with the

presence of raw, bleeding sores on the cutaneous as well as the mucous surface of the labia (see annexed Fig.). The temperature of the animals was not affected, and their general health appeared to be practically undisturbed.

Raw, ulcerating sores were still present inside the sheath of one of the rams, but the eruption on the upper lip of the other one appeared to be healing.

As the rams were marked with colour on the breast and between the fore legs, it was possible to determine which ewes had been tupped, and it was thus ascertained that one or two of them had contracted the disease although they had not been served by either ram. It may be observed that the ewes had been docked very close, and that the stump of tail left was quite insufficient to cover the vulva. The ewes were much tormented with flies at the time, and it appears to be possible either that some of them were thus infected, or that those which had not been tupped were infected by the disease on the nose of one of the rams.

The affected ewes were isolated, and the sores were dressed with mild antiseptics. Latterly, I dressed the lesions on the vulva with iodoform ointment. Under this treatment all the affected animals gradually recovered.

A CONTAGIOUS DISEASE OF THE GENERATIVE ORGANS IN SHEEP.

By J. M'FADYEAN, Royal Veterinary College, London.

HAVING had the opportunity to see the animals that were affected in the peculiar outbreak described by Mr Flook in the preceding article, I am induced to publish the notes of another outbreak of the same kind which had previously come under my notice.

Towards the end of September 1896 it was reported to me that a peculiar disease had made its appearance among a flock of ewes in the county of Suffolk, and on the 29th of that month a visit was made to the farm in question. It was then found that twelve ewes of the flock were all affected as follows: The labia of the vulva were intensely inflamed, swollen, tense, and painful. The lining membrane of the vulva showed inflammatory congestion, and in some cases ulcers were present either on the outside or the inside of the labia. There was a slight purulent discharge from the genital passages.

The explanation of the outbreak which naturally suggested itself was that the ewes must have been infected by a ram suffering from the same complaint. Unfortunately, it could not be ascertained whether that surmise was correct or not, as the ram which had been with the ewes was a hired one, and it had been returned to its owner six days previously. It was not even known with certainty that the whole of the twelve affected ewes had been served by this ram; he had had access to ten of them, but he had not been with the other two to the knowledge of the shepherd.

With a view to future experiments, pledgets of sterilised cotton wool were rubbed over some of the lesions so as to collect a little of the discharge, and on the following day one of these pledgets was

inserted into the vulva of a yearling ewe at the Royal Veterinary College. Another pledget was introduced within the sheath of a wether, and a third was inserted in the vulva of a cow. An hour afterwards the piece of cotton wool was found to have dropped out of the vulva of the ewe, and the plugs were then removed from the cow and the wether.

This procedure had entirely negative results in the ewe and the cow, but on 2nd October the sheath of the wether was found to be slightly swollen around the orifice. On the following day the swelling had increased, and a brownish crust or scab had formed on the skin close to the opening of the sheath.

On 4th October the sheath was still more swollen and reddened, and there was a slight discharge from it. It was now impossible to expose the penis. During the following ten days the swelling persisted, while the small sore which made its appearance on 3rd October continued to spread as a red ulcerating surface around the opening of the sheath.

On 15th October two small abscesses were found to have formed on the prepuce near the primary sore, and these on the following day had become converted into shallow ulcers. On the 16th a third small abscess had formed. On the 17th all the ulcers had increased in extent, and were covered with brownish crusts. After this date the sores began to heal, and had disappeared in about a fortnight.

On 14th October an attempt was made to transmit the disease by means of cotton wool and discharge from this wether to another wether and a ewe, but neither of these animals became infected. An attempt to isolate from the sores an organism capable of inducing the disease also failed, and the experiments thus came to an end.

It is scarcely possible that this can be a disease of common occurrence among sheep, as it is not one that can escape notice, and it must long ere this have attracted attention if it had been common. If the circumstances were favourable, the disease is evidently one that might spread indefinitely; but, since it seems to run a comparatively rapid course, and always ends in recovery, a flock infected with it one season would probably have become clean by the following season. There may, however, be exceptions to this rule, the disease being continued from one season to the next, either by a ram or a ewe that has not made a complete recovery. At anyrate, it would appear to be a not altogether needless precaution to see that newly-purchased rams are free from any sign of the disease.

A CASE OF EPIZOOTIC LYMPHANGITIS.

By J. M'FADYEAN, Royal Veterinary College, London.

HISTORY teaches us that great wars have always contributed to the dissemination of disease, both among human beings and among the lower animals, and it is already evident that the recent campaign in South Africa has conformed to this rule. By that agency glanders was spread throughout the whole of the Transvaal and Orange River Colonies, as well as in Cape Colony and Natal. That was only what might have been expected. What was not foreseen was that the

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transport of horses incidental to the war might also be the means of introducing a new equine disease into Great Britain. That, however, is what appears to have happened, for the disease called "epizootic lymphangitis," hitherto unknown with us, has now obtained a footing in this country.

It has been known for some time that since the close of the late war cases of this disease have been detected among army horses, but, with the exception of the animal figured in the accompanying illustration,



apparently no case of the disease has yet been discovered among horses belonging to private persons. It is scarcely to be hoped, however, that the disease is, with this exception, still confined to horses belonging to the army, and this particular case is here referred to mainly with the object of putting veterinary surgeons in private practice on their guard against the possibility of overlooking others of the same kind.

The horse figured here is a roan gelding which was brought by its owner to the College Free Clinique on account of the disease which affected its near hind leg. In the absence of my colleague, Professor Woodruff at the moment, I was asked to look at the animal, and I frankly admit that had my suspicions not been aroused by the fact that the patient was a cast army horse, I should in all probability have diagnosed the case as one of farcy, and detained the animal until it could be tested with mallein, according to the usual practice.

The owner stated that in the month of September last he had chased the animal at a sale of cast army horses, but this statement was not necessary to prove the horse's history, as he had the "blue arrow" branded on the near hind quarter, and had other brand marks on both shoulders.

The horse was in poor condition, and his near hind leg was swollen from the foot to the thigh. Between the coronet and the middle of the leg there were several discharging sores, and there was also a considerable raw ulcerating surface immediately above the hoof. The animal was very lame on this leg.

An unburst subcutaneous nodule on the inner side of the limb a little distance above the hock, was at once lanced, and some of the pus thus obtained was submitted to microscopic examination in the unstained state. By this simple means the nature of the disease was placed beyond any doubt, for the pus was found to contain great numbers of the so-called "cryptococcus." Subsequently the horse was tested with mallein, without eliciting the least reaction either as regards temperature or local swelling.

I do not propose to discuss fully on the present occasion the differential diagnosis of this disease, and, with regard to the naked-eye appearance of the lesions, I shall only remark that I am quite unable to recognise any macroscopic character of those present in this animal from which one could confidently diagnose the case to be not one of ordinary farcy. Fortunately, however, as already indicated, the correct diagnosis is easily assured when a little of the pus or discharge from the lesions is examined microscopically. A magnification of 500 suffices, but one of 1000 is better, and, for reasons that will be apparent immediately, there is little or no advantage in staining the cover-glass preparation. The best plan, in fact, is to place a speck of the pus on a glass slide, and apply a cover-glass with gentle pressure. The cryptococci are readily detected owing to their relatively large size, their oval outline, their bright refractile aspect, and their possession of a double-contoured envelope or capsule. The largest organisms measure about 4μ in their longest diameter, and the smallest are little more than half that size. In a preparation made from the pus of a lesion that has not burst spontaneously the majority of the cocci may be contained in the interior of the leucocytes, as many as four or five being present in one cell.

The cryptococci cannot be satisfactorily stained by any of the methods in general use for the staining of bacteria. Most of them are colourless even after twenty-four hours' exposure to carbol-fuchsin at ordinary temperatures, and the result is scarcely any better when the temperature of the stain is raised to the boiling point. It is for this reason that there is little or no advantage in staining films of pus containing the parasites; and, if preparations so treated are afterwards mounted in the customary manner in Canada balsam, the method has a positive disadvantage, inasmuch as the cryptococci are then less refractile, and the double contour of the envelope is less manifest. In a stained preparation, however, the unstained parasites may show up distinctly when included in stained cells, or when the albuminous film in which they lie has retained some of the colour of the stain. It was by taking advantage of this fact that Fig. 2 in Plate IV. was obtained.

The fact that epizootic lymphangitis has now been introduced into this country shows that it was an unwise procedure to bring back army horses from South Africa, and it was still more unwise to sell such returned horses to private persons. The horse here referred to had no sign of the disease for some weeks after he was bought by his last owner; but, in view of the long period of incubation which the disease is known to have, and the fact that many cases have occurred in the army since the early part of the year, there can hardly be a doubt that the animal was infected at the time of purchase. Some thousands of horses are known to have been spread about the country in this way, and the footing which the disease has thus obtained in Great Britain and Ireland may prove to be permanent.

FIVE CASES OF HODGKIN'S DISEASE IN THE LOWER ANIMALS.

By J. M'FADYEAN, Royal Veterinary College, London.

AT the present time it appears to be impossible to frame a precise definition for the terms Hodgkin's disease, lymphadenoma, and lymphoma, as they are applied in human pathology. The terms appear to be applied almost indifferently to any case in which there is, as a primary lesion, general or wide-spread enlargement of the lymphatic glands, provided this is not accompanied by a great increase in the number of leucocytes in the circulating blood, and provided no bacteria can be found in the enlarged glands. The first of these reservations excludes cases of lymphatic leucocythæmia, and the means of distinguishing between that condition and Hodgkin's disease are, of course, easily applied. On the other hand, it is by no means always easy to obtain assurance that the gland enlargement is not due to bacterial irritation. In general it may be said to be impossible while the patient is still alive, and it may involve a good deal of trouble after death. It is to be feared that a good many cases are set down as Hodgkin's disease without taking any trouble to exclude this possibility, except perhaps to see that no such gross alterations as suppuration or caseation are present in the enlarged glands. Obviously, unless particular care is taken to prevent it, a case of tuberculosis may be set down as one of Hodgkin's disease or lymphadenoma, and it is well known that until comparatively recently such mistakes were often made in connection with cases of equine and porcine tuberculosis. Some authors believe that similar mistakes have been made, and are still being made, in cases of glandular enlargement in the human subject.

One naturally asks whether the glandular enlargement which is the chief characteristic of Hodgkin's disease has not got a definite histology by which the affection may be readily distinguished from tuberculosis and other conditions in which numerous groups of lymphatic glands may be involved. The question has sometimes been answered in the affirmative. Two years ago this subject was discussed by the Pathological Society of London,¹ and on that occasion Dr Andrewes laid stress upon the following changes as

¹ "Transactions of the Pathological Society of London," Vol. LIII. p. 306.

distinguishing "true lymphadenoma" from tuberculous and other forms of lymphatic enlargement :—

1. A simplification of the structure of the gland, the distinction between cortex and medulla being abolished.

2. A diminution in the number of the leucocytes, so that they no longer conceal the framework of the gland.

3. A corresponding increase in the amount of the fibrillar reticulum or framework of the gland, this increase sometimes amounting to an actual fibrosis.

4. The presence in this hyperplastic reticulum of a peculiar type of endothelial cell, often of large size and multi-nucleated, but yet having little resemblance to the giant cells met with in tubercle. In view of their constancy in the disease, it was proposed to call these cells "lymphadenoma cells."

These views are quoted here, not with any idea of denying their accuracy with regard to lymphatic enlargement in the human subject, but to make it plain that, if the characters above-mentioned are essential to Hodgkin's disease or lymphadenoma, the cases which I am about to describe have no title to be called by these names. But, if the condition of which these cases are illustrative cannot be enrolled under the head of Hodgkin's disease, it is equally certain that they are not covered by any other recognised term, and that a new name is required for them.

The cases are five in number, four of them being in dogs and the fifth in a pig. They all came under observation during the present year. Unfortunately I did not see any of the animals alive, and in only one of the cases had I an opportunity to make anything like a complete *post-mortem* examination. That case I shall describe first.

Case I.—The subject in this case was an aged fox terrier, which was destroyed by Mr Henry Gray, M.R.C.V.S., Kensington, and forwarded to me for examination. The following are the notes made at the time of this examination :—

A considerable amount of fat is present under the skin and in the abdomen. The lymphatic glands are enlarged as follows. On each side of the penis (superficial inguinal) the glands are about the size of a hen's egg. Popliteal group about an inch in diameter. At the entrance to the pelvis on either side there is a gland as large as a hen's egg. The mesenteric glands are enlarged, forming a mass larger than a hen's egg. Bronchial glands about the size of large hazel nuts. In the anterior mediastinum there are two glands somewhat larger than that. Axillary glands about the same size as the popliteal. On the masseter, below the root of the ear, there is a gland about an inch in diameter, and behind and internal to that another twice as large. On section, all these glands present a grey or almost white surface. At some places the gland tissue is streaked red with small cut vessels and bleeding points. For the most part the tissue is abnormally soft, scarcely firmer than cerebrum. The spleen is enlarged, and its edges thickened and rounded; it weighs 14½ oz. On section, its pulp is maroon-red and beset with Malpighian bodies which are about 2 mm. in diameter. It also contains three pea-sized nodules, which in colour and consistence resemble the tissue of the enlarged lymphatic glands. Consistence of the splenic tissue rather soft. Both kidneys are reduced in size, and weigh 1 oz. each.

The capsule is adherent and the surface irregular. Renal tissue tough (chronic interstitial nephritis). Liver, heart, lungs, and thyroid glands normal. Stomach and intestines not sent. Smears of blood which had been made by Mr Gray during life, and others made by me after death, showed no excess of leucocytes.

Although both the appearance and the distribution of the lesions contra-indicated tuberculosis, it was thought well to inoculate two guinea-pigs with an emulsion made by rubbing down a portion of one of the enlarged glands with sterile water. The inoculation was subcutaneous, and the animals were found to be perfectly healthy when they were killed four months afterwards.

Case II.—The animal in this case was a Newfoundland dog, and the only parts sent to me were the spleen and a number of lymphatic glands. The latter presented exactly the appearance noted in connection with the preceding case. The spleen weighed 1 lb. 13½ oz., and for the most part the normal tissue of the organ had been replaced by a new tissue of the same white, marrowy character as the glands.

Case III.—In this case I received only one large lymphatic gland and the spleen, which was in a similar condition to the spleen of Case II. Age and breed of the dog not known.

Case IV.—The subject in this case was a Whippet bitch, three years old. Only five glands were sent—two being from the region of the throat, two others bronchial, and the fifth mesenteric. The throat glands were each as large as pigeon's eggs, the bronchial somewhat smaller, and the mesenteric was about the size of a hazel nut. All these glands had the same appearance as those of Case I. The spleen was not sent, but it was said to have been enlarged and firm. The animal had been ill and under treatment for three months before it died.

Case V.—The animal in this case was a pig. The lymphatic glands were said to have been generally enlarged, and those sent to me were whitish in colour and almost cerebral in softness. A piece of liver and a few inches of a rib were also sent. The former contained a number of soft, white, tumour-like growths, and tissue of a similar appearance had at one part taken the place of the osseous substance of the rib.

Histology of the Lesions—The lesions in all these cases were found to be of a remarkably uniform character. The nature of the alterations is shown in Figs. 3 and 4 in Plate IV. At the outset it may be said that the alterations were entirely different from those laid down by Dr Andrewes as characteristic of Hodgkin's disease in the human subject, save that in the lymphatic glands the distinction between cortex and medulla was altogether abolished. The coarser strands of connective tissue and the finer reticulum of a normal lymphatic gland had almost entirely disappeared, and a section from any part of the diseased glands showed scarcely any histological element except cells of the lymphocyte type, and an occasional capillary. Epithelioid cells were conspicuous by their absence. The white marrowy tissue of the enlarged spleen in Cases II. and III. had an identical structure, and, save for the presence of remnants of the splenic trabeculæ, sections from these spleens could not be distinguished from sections from the enlarged lymphatic glands.

DESCRIPTION OF PLATE IV.

FIG. 1. To illustrate the article by Mr Riddoch, p. 357. Micro-photograph ($\times 1000$) showing tubercle bacilli in expectorate from a cow. The preparation was stained by the Ziehl-Neelsen method.

FIG. 2. To illustrate the article on Epizootic Lymphangitis, p. 376. Micro-photograph ($\times 500$) of pus from the case described in the article. The line *a* is drawn from a cell, which contains two cryptococci, showing as unstained, vacuole-like, oval spots in the cell-substance. The lines *b* and *c* are drawn from cells which contain several cryptococci. Elsewhere in the figure a number of other cells also containing cryptococci are seen less distinctly.

FIGS. 3 and 4 illustrate the article on Hodgkin's Disease in the Lower Animals, p. 379. Fig. 3. Micro-photograph ($\times 250$) of a section from the spleen of Case II. Fig. 4. Micro-photograph (500) from one of the enlarged lymphatic glands of the same dog.

HODGKIN'S DISEASE IN A PIG.

By J. F. HODGSON, M.D., Ch.B., Vict., Assistant Medical Officer of Health of the County Borough of Halifax.

Very little is known concerning the pathogeny of Hodgkin's disease, and as yet experimental investigations have not resulted in anything satisfactory. It is thought by some to be related to tuberculosis, so that the occurrence of Hodgkin's disease in a pig—an animal in which tuberculosis has a great tendency to become generalised—may be perhaps more than a coincidence, and is of more than usual interest.

In this case the pig was the smallest of a litter killed in the Halifax public slaughter house. It weighed from 120 to 140 lbs., and its appearance before it was killed called for no comment either from the buyer or the slaughterers.

There was a general enlargement of all the lymphatic glands in the body; some were as large as a hen's egg. They were greyish-white on section, and of the same consistence as normal lymphatic glands. There were no adhesions, the glands being freely movable one upon another, and no caseation or suppuration. The liver weighed 3 lbs. 10 ozs., was slightly enlarged, and had a number of yellowish-white tumours, some of which measured as much as $1\frac{1}{4}$ in. in diameter, scattered throughout its substance.

The spleen did not appear to be enlarged, but the Malpighian corpuscles, being rather larger than normal, were rendered very distinct. Some soft flat masses of lymphoid growth were situated between several of the ribs and the pleura. The bone marrow had the appearance of a reddish-grey mucilaginous substance.

Microscopically the smaller glands showed simply an excess of lymphocytes above their normal structure, while in the larger glands this excess was very much more marked, and the reticulum appeared much finer. The growths in the liver seemed to originate in the interlobular spaces, and in appearance resembled that seen in the glands. There was no opportunity to examine the blood, but it was noticed that the flesh was paler than that of the rest of the litter. This was an advanced case, and so rendered the diagnosis easy.



Fig. 1.

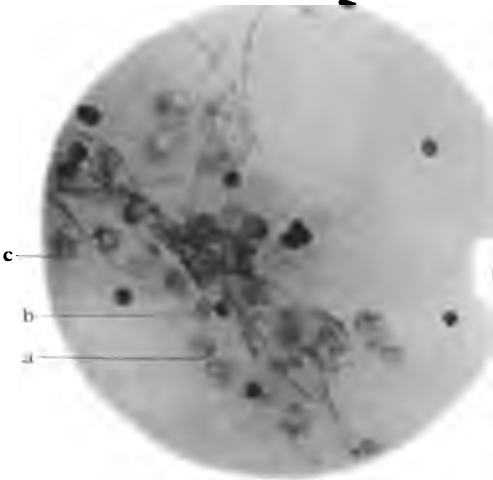


Fig. 2.



Fig. 3.

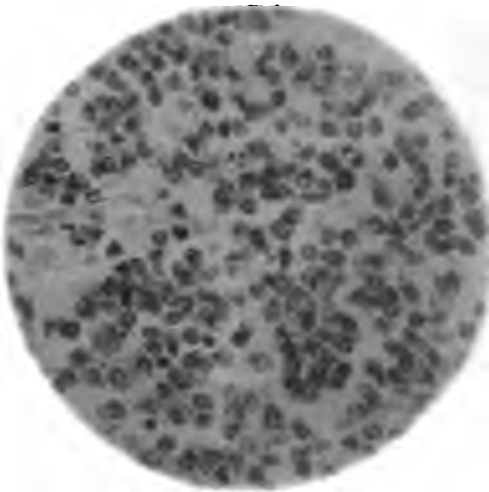


Fig. 4.

Abstracts and Report.

THE IDENTITY OF SWINE ERYSIPELAS AND URTICARIA IN THE PIG.

By H. SCHMIDT.

On the basis of bacteriological evidence Jensen and Lorenz have already come to the conclusion that the organism of swine erysipelas is identical with that of urticaria. This now generally acknowledged fact is supported by the following interesting case.

On the 10th September a sow far advanced in pregnancy showed a body temperature of 41.8°C . Later the animal exhibited great weakness, particularly of the hind quarters, and practical inability to stand. Appetite was completely in abeyance, and the skin covering the back and belly showed round red spots of varying sizes and in part confluent.

Treatment consisted in the injection of about thirty-five cubic centimetres of swine erysipelas serum, the injections being made on the 10th, 12th, and 13th September.

On the 14th September the sow farrowed with a litter of eight, which appeared quite healthy to the owner. On the 15th the whole of the eight little pigs showed about two to three sharply-defined, rectangular, bluish-violet, somewhat elevated spots, from one to two centimetres in circumference. Clearly they were suffering from urticaria.

The reddish patches on the sow had partly disappeared, and were more concentrated towards the neck, where they formed a dark bluish-red patch about eight inches in diameter. The animal again showed loss of appetite. Each of the little pigs received one cubic centimetre of lymph. One died; the others developed well.

The simultaneous appearance of disease amongst all the one-day-old pigs seems only capable of the one interpretation, viz.—that infection had already occurred during foetal life, and that the serum treatment of the sow had caused weakening of the virulence of the swine erysipelas bacilli, so that the latter were only capable of producing the milder form of swine erysipelas, i.e. urticaria.—(*Berliner Tierärztl. Wochens*, 28th May 1903, p. 351.)

GUTTUROMYCOSIS IN THE HORSE.

By J. N. RIES.

IN 1873 Rivolta had under observation two horses suffering from a disease which he was the first to describe, and which he termed gutturomycosis. In 1881 Bassi saw the same disease in a horse and a mule. The Italian authors considered the disease due to a parasitic fungus which they termed *gutturomyces equi*. It has been suggested that the lesions observed were of a glanderous nature. The following case tends to support the theory that gutturomycosis as described by Rivolta actually occurs.

The subject was a gelding, aged 12 years, which had several times changed owners. Eighteen months before the appearance of the first symptoms of gutturomycosis, and whilst in the hands of a previous owner, the horse had

fallen in the shafts and had fractured several rings of its trachea. During last year it suffered from two mild attacks of hæmoglobinuria. Without apparent cause it developed a discharge from both nostrils, which caused it to be suspected of strangles. The horse's throat had been poulticed, an electuary given, and clothing applied. This treatment was continued for ten days, but, as the horse fell away considerably, Ries was consulted.

The eyes were retracted into the orbits. The depression above the orbit was very marked. The animal was tucked up, and showed the sub-costal depression suggestive of pleurisy. The manner in which the head was extended, and the extent of the flank movements, suggested some difficulty in respiration, but no fever or serious disturbance of the chief functions could be detected. The lips were dry, and there was no discharge of saliva from the mouth, but an abundant discharge mixed with particles of food ran from both nostrils. At short intervals the patient tossed its head and sneezed, ejecting large flakes of perfectly chewed food mixed with saliva. When water was offered it greedily attempted to drink, but immediately swallowing commenced water escaped from the nostrils, whilst weak peristaltic movements flitted downwards along the jugular furrow, and bubbles of gas appeared to be rising in the opposite direction. By applying the hand to the œsophagus an ascending wave which alternated with the movements in the opposite direction could be detected. As soon as these swallowing movements ceased the œsophagus became relaxed, and was then almost indistinguishable from the carotid, except by its failing to exhibit pulsation. For a minute the animal continued its attempts to swallow, then stopped drinking, and extended its head. The lips and the cheeks were retracted, the skin covering them became wrinkled, and that in front of the trachea exhibited longitudinal folds; a swelling passing from below upwards appeared in the œsophagus, and attempts to vomit, followed by coughing, ended in the animal rejecting through the mouth and nostrils all the liquid taken, together with fragments of vegetable material and pellets of food. In a few minutes a fresh attempt to drink was made.

The above-described phenomena were repeated five or six times. Each time the food returned was less in quantity, but some solid material, particularly bran, was always present. The nostrils, having been cleared by repeatedly administering water, were examined, but revealed nothing abnormal. The breath was neither fœtid nor smelt of chyme. The sub-maxillary region was free of swelling; the parotid was slightly shrunk, but appeared painless on pressure. The first ring of the trachea exhibited an irregular callus, the fractured ends of the rings overlapping. The next two rings in the lower third of the neck were similarly fractured. The animal violently resisted attempts to withdraw the tongue from the mouth, and this resistance was very remarkable, considering the general state of depression, and the fact that the effects of starvation were beginning to make themselves felt.

Diagnosis vacillated between stricture of the œsophagus, stenosis, impaction, and tumour-formation. Hot moist compresses were applied to the throat and neck; milk and cold water *ad libitum* were ordered; nutritive enemata were given, and solid food forbidden. Two days of this treatment seemed to be followed by improvement. The owner said the water and milk were not returned.

During a second visit hay was offered. The animal took a mouthful greedily, but chewed slowly and with difficulty. It was better able to masticate green food. It had succeeded in eating a large handful without returning any by the nose, and without producing spasm of the œsophagus, when suddenly a violent attack of nausea accompanied by the peculiar curling of the nostrils above mentioned came on, and all the food which had

been swallowed, including the hay, was returned by the mouth and nose. After this Ries considered himself justified in regarding the case as spasm of the pharynx and œsophagus.

On examining the chest some râles were detected in the lower part of both lungs. Mechanical pneumonia appeared imminent, and in fact set in twelve days after the first signs of spasm of the œsophagus.

Believing that the only way to check the course of this pneumonia was to prevent further infection of the lung, Ries performed œsophagotomy. An elastic sound was passed in the direction of the stomach to a distance of twelve inches, at which point it encountered opposition. Gentle pressure caused the animal great pain. In the direction of the pharynx the sound passed readily. Ries contented himself with this intervention.

On the fifteenth day septic pleurisy appeared. The animal showed intense thoracic pain, which it exhibited by turning the head from time to time towards the sides. The pulse became imperceptible, and vague colicky attacks were soon followed by involuntary micturition: the animal was dying. To facilitate carrying out a *post mortem* examination under the most favourable conditions, the horse was killed by dividing the femoral artery.

The small intestine contained an almost limpid, lemon-yellow liquid; the stomach, three to four quarts of a similar liquid bearing in suspension a few fragments of food. The mucous membrane of the right sac presented the iridescent red colouration seen in starvation. The wall of the left sac showed œdematous infiltration about one and a half inches in thickness over the cardia, an infiltration which entirely effaced the true structure of its three layers. The lower portions of both lungs were the seat of septic gangrenous hepatisation; in addition, the lesions of septic pleurisy could be detected. The upper two-thirds of the lungs were healthy. The œsophagus was normal throughout its entire length. The trachea showed the fractures already noted during life.

The mucous membrane of the larynx and trachea was soiled with particles of food, and appeared of a dirty grey colour. On the inner wall of the left guttural pouch was a sharply-projecting, white, velvety patch formed by a fine growth of a very pure mycelium. It was elliptical in shape, the greater length being vertical; it measured $2\frac{1}{4}$ inches by $1\frac{1}{2}$ inches, and was adherent over the insertion of the rectus capitis. On removing this mycelium a thin black pellicle was found, which, when detached, revealed a yellowish-white magma, fairly firm in consistence, and forming almost the entire thickness of the growth. The rest of the guttural pouch was absolutely healthy. The mucous membrane presented a shining appearance, and the entrance to the Eustachian tube was closed.

Microscopically examined, the mycelium appeared entangled and exhibited dichotomic divisions. Cultures proved it to consist of *aspergillus fumigatus*. Sections through the thickness of the lesion showed this to consist chiefly of an exudate surrounding four nerve bundles in a profound state of degeneration: the glosso-pharyngeal, the pharyngeal branch of the pneumo-gastric, the hypoglossal, and a part of the superior cervical ganglion. On microscopic examination the nerve fibres were found partly degenerated and invaded by a connective-tissue new growth, as well as numerous radiating tufts of mycelium.

It should be mentioned that the vegetating growth was entirely in relief, that it exhibited none of the characters of an ulcer, and that it differed essentially from a glanderous lesion. Some cultures produced the *aspergillus fumigatus* in a state of purity, and others a *staphylococcus* in addition. The bacillus of glanders could not be detected either microscopically or by making potato cultures.

In the absence of inflammatory appearances about the organs of the mouth, the hyperæsthesia of the tongue and the difficulty in masticating suggested

some disturbance of innervation of the tongue. The spasm of the pharynx and œsophagus and the reflex closure of the cardia might be referred, in the absence of anatomical lesions of these organs and of respiratory and cardiac disturbance, to some change in the pharyngeal and œsophageal branches of the pneumo-gastric. The failure to detect brain disturbance forces one to reject the idea of any lesion in the cerebral centres of the affected nerves. Injury at the point where the hypoglossal, pharyngeal, and the œsophageal and pharyngeal branches of the pneumo-gastric have structural relations or come together could alone explain the symptoms observed, and this point is in the guttural pouch.—(*Recueil de Médecine Vétérinaire*, 15th April 1903.)

CYTOTOXINS.

By M. KAUFMANN.

The blood is a liquid medium which bathes all the living elements of the organism, furnishing them with the materials necessary for their nutrition and function; receiving from them, on the other hand, various products, the result of wear, degeneration, and secretion. The greater part of the materials thus returned to the blood by the cells of the tissues play no particular part. They are only destined to be excreted, principally by the kidneys and lungs. Among these products, however, are some which play an important physiological part. The secretion of certain cells is necessary to the regular performance of function by other forms of cells, and when these products are absent disturbance at once occurs in the functions indicated.

Thus, for example, the absence from the blood of the special material secreted by the pancreatic cells brings about that serious disturbance in nutrition known under the name of saccharine diabetes. Similarly, removal of the thyroid and parathyroid glands produces myxœdema, cretinism, and other disturbances, varying according to the species of animal in which the operation is performed and various other conditions. We also know, according to Claud Bernard, that the hepatic cells continually furnish sugar to the blood passing through the liver, sugar which is indispensable for the nourishment of all the other cells of the organism.

These examples are sufficient to show that in addition to the part the blood plays as a nutritive and as an excretory liquid it exercises that of exciting or sustaining certain functions or of regulating them by means of special secretions produced by certain cells.

The organs of one and the same individual reciprocally act and react through the medium of the nervous system and of the blood.

The influence exerted at a distance by means of the blood stream is often more important than that transmitted by the nervous system, but most commonly the transmission is double, that is, it occurs through both the nervous and blood channels, and is in consequence more perfect.

I.

Even in the same animal or individual, the blood if comparatively examined in different tissues has not at all points the same properties. Each tissue communicates to it a particular quality. In any two individuals the constitution of the blood is different, and the difference is greater the further apart the two individuals happen to be in the zoological scale. The blood draws its special qualities from the products secreted by the living cells which it bathes. Now, the different kinds of cells secrete products which are by no means identical, and which usually differ among themselves.

It results from the preceding that the blood of a given animal may have

injurious and even toxic properties for certain of its own cells, or, and still more frequently, for the cells of another animal. These products of cellular secretion which render the blood of an animal toxic for certain cells have been given the names of cellular poisons or cytotoxins. The cytotoxins, therefore, are poisons of animal origin, which are poured into the blood and communicate to it the power of killing certain cells.

To clearly demonstrate the presence of cytotoxins in the blood a quantity of blood is allowed to coagulate, sheltered from germs, and the serum so obtained is caused to act either *in vitro* directly on isolated living cells like microbes or blood parasites, or is injected into the veins, under the skin, or into the peritoneum of an animal, and so allowed to act on the entire organism.

Certain microbes have difficulty in developing, or fail altogether to develop, in blood serum; while others may even be killed by certain sera. These toxic sera are therefore termed microbicidal or bactericidal. This bactericidal power of the serum assists the animal in defending itself against microbic infection, and plays an important part in natural or acquired immunity against infectious disorders. In order to study the cytotoxins in blood serum one usually uses as a reagent the red blood corpuscles, that is to say, the living normal elements of the blood.

We know that in blood removed aseptically from the vessels of an animal the red blood corpuscles undergo no alteration; they preserve their form, colour, consistence, and all their normal characters. But if, instead of adding to this serum red blood discs from the animal which has furnished the blood, we add to it some obtained from another animal, particularly from a different species, the red blood discs may undergo change and be rapidly destroyed. There is, therefore, in the serum a substance capable of destroying certain red blood corpuscles, a hæmolysant substance, a special cytotoxin which dissolves certain red blood corpuscles derived from an outside source.

The normal blood of an animal is not toxic for its own red blood corpuscles. Usually it does not injure the red blood corpuscles of individuals of the same species, nor, it may be, of species distinctly different; but a blood which is normally not hæmolytic for the red blood corpuscles of any given animal may become so experimentally.

Bordet has drawn attention to the artificial production of a cytotoxin in the guinea-pig's blood by the following experiment. Having discovered that the serum of a normal guinea-pig's blood does not affect the red blood corpuscles of the rabbit, or scarcely so, he afterwards tested the effect on these same red blood corpuscles of the rabbit of serum obtained from a guinea-pig which had for some time previously been given subcutaneous injections of rabbit's blood. He found that the serum of the prepared guinea-pig, that is to say, the guinea-pig which had undergone several injections of rabbit's blood, dissolved the red blood corpuscles of the rabbit with remarkable energy. Thus, under the influence of hypodermic injections of rabbit's blood, the cells of the guinea-pig's body had secreted and poured into its blood a hæmotoxin or hæmolysin destructive to the red blood corpuscles of the rabbit.

Bordet has shown, moreover, that this special hæmotoxin produced by the guinea-pig's cells and poured into its blood is formed of two different substances, one of which, the alexin or cytase, is destroyed by heating to 56° C., whilst the other, the sensitising substance or the phytocytase, resists this temperature perfectly, and is not destroyed before 66° C.

Neither of these two substances have any manifest hæmolytic action when alone. A serum which only contains one of them, either alexin or phytocytase, is not hæmotoxic.

Alexin or cytase exists in all normal serums, whilst the sensitising substance is not found in these serums, but always appears in animals pre-

vously treated by injections of blood. The serum, therefore, is active when it contains the two indispensable substances. The serum of a fresh subject which only contains alexin, and which is inactive as regards the red blood corpuscles, may become very hæmolytic if one adds to it a certain quantity of serum from an animal prepared by injections of blood. When this addition has been made to new serum the latter is said to be active.

The discovery of hæmotoxin and of a method of producing it artificially has opened the way for a large number of researches, which are now being carried out, particularly in two directions. On the one hand, investigators are attempting to produce cytotoxins active against various cells, like spermatozoa, hepatic cells, renal cells, nerve cells, etc. On the other hand, an attempt is being made to discover the mode of action of the cytotoxins on their corresponding cells.

II.

Metchnikoff succeeded in producing a serum against leucocytes, and another against spermatozoa. Landsteiner, of Vienna, obtained about the same time a serum spermatoxic for the spermatozoa of the bull by injecting the semen of this animal into rabbits. The serum of the rabbit thus prepared rapidly paralysed the spermatozoa of the bull.

Metchnikoff, by injecting rabbits with the pulp obtained by crushing the lymphatic ganglia of the same animal, obtained a serum which had the power of destroying the white mononuclear and polynuclear blood corpuscles of this rodent. Added in small quantities, this serum almost instantly immobilised the leucocytes of rabbits and brought about their destruction. This serum, therefore, might be termed leucotoxic.

Von Bungen has likewise obtained a serum which stops the movements of vibrating ciliæ by injecting mucus from the trachea of the ox into guinea-pigs.

Lindemann, by injecting the renal substance of the rabbit into guinea-pigs obtained from the blood of these guinea-pigs a serum which, when injected into rabbits, produced albuminuria and symptoms of acute nephritis. This serum therefore was toxic for the kidney cells of the rabbit, and was in fact nephrotoxic.

Delezenne and Deutsch have prepared a hepatotoxic serum for the hepatic cells of one species of animal by injecting the liver substance of the same species into rabbits or guinea-pigs. Delezenne and Metchnikoff have also attempted to produce a neurotoxin—that is to say, a poison for the nervous cells. They succeeded by injecting the substance of the dog's nervous centres into the peritoneal cavity of ducks. The introduction of a very small quantity of the serum of birds thus treated into the cerebral hemispheres of dogs killed them almost instantly.

From these facts one can deduce a general conclusion—*i.e.*, that the injection of any variety whatever of cells under the skin or into the peritoneal cavity of an animal provokes the production in the blood serum of that animal of a cytotoxin specific for the cells of the species of animal which has furnished the material for injection.

III.

Cellular poisons, as has just been shown, can easily be produced by injecting animals with cells obtained from other species. But the question arises if the resorption of the blood or cells of the animal itself is capable of producing in its blood cytotoxins for the red blood corpuscles or for its own cells. We know that blood extravasations and pathological exudates are readily absorbed, and that in different diseases certain tissues become atrophied or disappear by resorption. Is there then in these cases a formation of cytotoxin?

Several experimenters have endeavoured to solve the question.

Ehrlich and Morgenroth injected goats with goat's blood previously treated with water, in order to destroy a certain number of the red blood corpuscles. They thus obtained a serum which dissolved the red blood corpuscles of goats other than those which supplied the serum. In this case, therefore, there had been formed a cytotoxin which acted not merely on the red blood corpuscles of a strange species but even on the red blood corpuscles of the same species. The authors term this special form of cytotoxin, isotoxin.

Nevertheless, it must be remembered that this isotoxin is not an autotoxin, for it has no solvent action on the red blood corpuscles of the goat which has undergone the injections. The serum of that goat is only toxic for the red blood corpuscles of other goats.

Metelnikoff, under the direction of Metchnikoff, has endeavoured to produce not an autohæmotoxin but an autospermatoxin by injecting male guinea-pigs with semen from the same species. At the end of a short time the serum of animals thus treated immobilizes the spermatozoa of guinea-pigs in a few minutes. This toxic action occurs not only in connection with the spermatozoa of strange guinea-pigs, but also in connection with those of the guinea pigs submitted to the injection of semen. Here then is a case of a true auto-cytotoxin produced as a consequence of resorption of the cells of the same species. A very curious fact is that the spermatozoa of a guinea-pig whose blood serum is very spermatotoxic live without difficulty in the genital organs of the same animal, but are killed when mixed *in vitro* with a little of the same individual's serum. This difference of action is explained by the fact that spermatotoxin, like all the cytotoxins, appears to be formed of two substances, one of which circulates in the blood, the other existing in the bodies of the leucocytes, but not becoming diffused in the plasma as long as the leucocytes undergo no change. Now, this change would occur after removal of the blood, the leucocytes would then pour their cytase into the serum in which the phytocytase already existed, and the cytotoxin would become complete and active.

We see, therefore, that an organism may develop an autotoxin of which the two constituent parts remain separate and consequently inactive in the living animal. But it is possible to imagine that in consequence of some pathological change or other the leucocytes might undergo alteration and allow the cytase, which they had formerly contained, to escape; an active autotoxin would then be produced, for the two substances would be acting together, and a veritable auto-intoxication might result.

To sum up, the preceding facts show that under the influence of certain conditions the animal body manufactures particular poisons which have a specific toxic action on certain cellular elements. These poisons or cytotoxins, whether we are dealing with hæmotoxins, leucotoxins, nephrotoxins, hepatotoxins, spermatotoxins, neurotoxins, or others, are always formed by two different substances, which may remain isolated and then have no action, or may become mixed and then acquire the specific toxic powers of the corresponding cytotoxin. One of these elementary substances, phytocytase, may circulate in the blood plasma, the other, the cytase, remaining enclosed within the leucocytes. These two substances have the characters of true digestive ferments.

The study of cytotoxins is extremely interesting and important both as regards general pathology and therapeutics. The results it has given explain a number of intoxications, the mechanism of which has hitherto appeared extremely mysterious; and the best examples of which perhaps are uræmia and eclampsia.—(*Recueil de Médecine Vétérinaire*, 15th May 1903.)

PROFESSOR KOCH ON RHODESIAN REDWATER OR AFRICAN COAST FEVER.

THIRD REPORT.¹

IN continuing the record of the African coast fever investigation, it will be remembered that in my Second Report I explained that while a single injection of a susceptible animal, with blood drawn from an animal in the acute stage of the disease, did not reproduce the disorder in its characteristic form, repeated injections with such blood appeared to induce a mild attack characterised by an elevation of temperature, and the appearance of a certain number of parasites in the circulating blood. From this I surmised that there was a possibility of a certain degree of immunity being established by such successive inoculations, the extent whereof could only be gauged by submitting such inoculated animals to some method of infection severe enough to induce, in unprotected animals, an attack of African coast fever of an intensity similar to that ordinarily communicated by natural veldt infection.

The discovery of such a means of infection was greatly to be desired in order that we might be able to test the serum which we had prepared, and to decide certain other questions of importance. Various experiments were made to achieve this end.

The mild infection induced by repeated injections of blood taken from sick animals was passed through a series of healthy cattle, in order to determine whether the virulence of such attacks might not be heightened by such a transfer; but passage from one animal to another through five animals in no wise intensified the severity of the attack, the last animal reacting no more severely than the first. This experiment, however, proves that under certain conditions the organisms of coast fever may multiply and reproduce themselves in the blood of artificially infected animals, even when they give rise to no visible indications of disease. Our experiments have also shown that inoculations with the blood of recovered animals, which only contains an inconsiderable number of single parasites, will induce similar modified attacks of African coast fever, and, while these experiments have not been numerous, they tend to indicate that recovered animals are even more suitable for inoculation purposes than those which are actually sick.

In other directions we sought for a means of communicating the disease in its virulent form. For instance, intraocular injections with infected blood were tried without effect, and we also endeavoured by means of tick infection experiments to imitate natural methods. For this purpose cultures of the various varieties of suspected ticks were prepared. At first much difficulty was experienced in hatching out such cultures on account of the coolness and dryness of the atmosphere, conditions which experience has shown are unfavourable for work of this description. Ultimately, however, by the use of an incubator, in whose interior the humidity of the air was artificially increased, the eggs laid by ticks collected from our animals were hatched out as expeditiously as they are under the most favourable natural conditions, but when the young ticks so hatched were placed upon healthy animals, we found that, with the exception of certain doubtful cases, we failed to produce a characteristic attack. Trials were made with broods of various varieties of ticks—with broods hatched out at different temperatures and with broods kept for various periods before being placed upon the animals—and this work is still being continued. To approach natural methods still more closely, broods of young ticks were liberated in various localities on the grass, and

¹ The Report is dated 25th September 1903.

susceptible animals were subsequently grazed in such places. That this method should be successful appeared somewhat doubtful, as we expected that the drought, high winds, dust, and sun would speedily destroy the liberated ticks, but, in spite of the unfavourable weather, these larval broods remained where they were placed, being most abundant on the sheltered side of the grass stems away from the sun, and particularly plentiful at the extremity of the stalks, where they clustered together in small clumps apparently waiting for the passage of a suitable host to whom they might attach themselves. These larval ticks displayed no tendency to migrate or travel from place to place, but remained where they were placed for several months. High winds seemed to scatter them a little in the direction in which the wind was blowing, but no other atmospheric change appeared to affect them. Soon after sowing these broods of seed ticks in the veldt we found that it became highly infective. Previously only occasional cases of African coast fever had occurred amongst animals grazing in these places, the natural veldt infection appearing to be so slight that animals might graze there for many weeks without sickening, while latterly ticks had become exceedingly scarce and cases of sickness had been correspondingly few in number, apparently on account of the cold and drought.

The change in the infective property of the veldt was presaged by a gross tick infection of all our animals, many nymphs, and subsequently many full-grown ticks, were found upon them, and every susceptible animal which we exposed speedily became sick. Since then on several occasions animals have been turned into this veldt to test their immunity, together with a number of susceptible cattle to act as controls, and in each instance the controls became infected and died in about a month. From this circumstance we can safely assume that any animal remaining healthy upon such a veldt must be immune.

In this way we have attained the end for which we sought, and now have at our disposal a certain means of testing the immunity of any animal by a process which is preferable for our purpose to any other, because, after all, it is veldt infection which animals must be brought to withstand, and any artificial method of infection would ultimately have to be compared with and tested by veldt infection in order to prove its reliability.

Now, being in a position to test our animals from the first by natural infection, our experiments should satisfy all legitimate requirements.

I will now proceed to show what has been the result of testing our various experimental animals upon such an infected veldt.

These experimental animals may be divided into three groups, of which we will take, first, those animals treated with injections of blood.

A certain number of animals were subjected to a single inoculation with a small dose of blood taken from a sick or a recovered animal. These animals appeared to possess no immunity, as they all sickened and died; nor were animals injected with a single large dose of blood drawn from a sick animal in any better case, as 500 cc. of blood so given afforded no protection. Repeated injections were more satisfactory, and appeared to confer an undoubted immunity. Successive injections with blood taken from sick animals in doses of from 200 to 2000 cc., with an interval of from ten to twenty days between the injections, produce a high degree of immunity, which may be attributed to the fact that the blood of animals so fortified acquires properties which render it unsuitable for the multiplication of the specific micro-organisms of the disease. It is to be regretted that such a method of inoculation cannot be profitably employed on a large scale for protective purposes, on account of the difficulty experienced in obtaining blood in the quantity necessary for such a method of immunisation.

The effect of inoculation with smaller doses of such blood is not so

satisfactory, but I believe that repeated small doses of blood will confer an immunity which will be heightened in direct proportion to the number of injections to which the animal is subjected, and our experiments show that it is not necessary to employ the blood of sick animals for this purpose, as blood taken from recovered animals has a similarly satisfactory effect, and in some cases even a better one.

While the number of animals which we have treated in this way is at present small, and I cannot give figures to show what percentage of animals have been protected, all the work done promises so well and points so clearly to the establishment of an undoubted immunity, that I think the time has arrived when this method may be put to actual test in the field.

Of our experiments in this direction I may instance two characteristic examples.

The eight animals previously mentioned subjected to repeated blood injections, in the course of which the specific organisms of the disease were conveyed from one to another in the expectation that the disease would gain virulence in its passage, were afterwards sent into the infected veldt. One of these sickened and died after the usual interval, many parasites being present in the circulation: this animal appears to have had no immunity whatever. Numbers two, three, and four, sickened and died after a considerable interval, and in their case the disease underwent a marked modification, being characterised by an unusually small number of parasites in the circulating blood. For three months after it was turned into the infected veldt, number five continued perfectly healthy; it then became sick, and now only numbers six, seven, and eight survive. These are quite well to-day and appear to be immune.

A remnant of three out of eight is small, but allowance must be made for the fact that these animals only received two injections of the mildest virus at our command, therefore I consider this result may be taken as showing the minimum ratio likely to be protected by repeated blood inoculations. In the case of number five, and also, perhaps, in the case of two, three, and four, permanent protection would probably have resulted had the blood injections, by which they were temporarily immunised, been repeated.

A somewhat similar experiment was made with the blood of a cow which came from Beira, shortly after the establishment of the Hillside Station, and which can be considered immune, as it has been running on infected veldt ever since its arrival. In its blood we find the occasional coast fever parasites usually present in such cases. Two susceptible animals were infected subcutaneously with 20 cc. of this cow's blood on six successive occasions, with an interval of three days between each inoculation, and were afterwards turned out on the infected veldt. After some months one animal sickened and died. In this experiment I consider that the interval allowed between the injections was too short, and with more lengthy intervals results would have been better.

At present it is impossible to say definitely how long immunity of this description will last, how many injections should be made, or what space of time between the inoculations is best; nor can I state what class of recovered animals are most suitable for inoculating from.

For the solution of these and other questions experiments are still being made, but at present I am of opinion that the best results will be obtained by using freshly drawn defibrinated blood from recovered animals which are in good condition. This blood should be injected subcutaneously into each animal which it is desired to protect in a dose of 10 cc., and the injection should be repeated four times with an interval of seven days between each injection. Afterwards 10 cc. doses should be given for some time every two weeks, and later a dose once a month should suffice.

Continuing the record of our investigations, I now come to the second experimental group. In this are comprised animals treated with serum.

The serum used was prepared as I explained in my former report, some of our immune animals being injected with increasing doses of blood taken from sick animals, beginning with doses of 5 cc. and finishing with a maximum of 2000 cc., while others received a succession of doses of 2000 cc. each of sick blood injected subcutaneously, or of 1000 cc. injected intravenously. As successive large injections gave the best results, the system of gradually increased dosage was abandoned. In fortifying these animals, special care was taken to select samples of blood containing a large number of organisms, no blood being employed which showed on microscopic examination less than an equal number of parasites and blood cells. This precaution was necessary, as it was our intention to prepare a serum which would exert its influence specially upon the disease-producing organisms present in the blood.

After three or four large injections given at intervals of from two to three weeks, which were well borne by all the inoculated animals, the serum of these animals was found to possess very remarkable properties. When injected into healthy animals in doses up to 150 cc. no systematic disturbance resulted, but when sick animals were similarly treated, its administration was followed by a striking change in the African coast fever parasites circulating in their blood. The parasites became smaller, their outline was lost, sometimes they were scarcely visible, and in the course of a few days they disappeared. Carefully-kept records show that in every case in which serum was administered, even to animals which were highly infected, there was always a marked reduction in the number of parasites, and sometimes they vanished altogether. Details of these records, which possess a high scientific interest, I hold over for a later and more exhaustive report. This specific action of our serum upon the organisms of the disease was exactly what we sought, but we found that, unfortunately, it also possessed, in a very high degree, an undesirable hæmolytic property which exerted a solvent action upon the blood cells of sick animals. A slight hæmolytic action was looked for as the inevitable result of the association of red blood cells with the organisms in the blood used for fortification, from which it was impossible to separate them, but the exceptional character of the hæmolysis produced in sick animals inoculated with this serum, while healthy animals remain unaffected, would indicate that in this disease the red blood corpuscles are in an unusually unstable condition.

Injection of 50 cc. of well prepared serum into a sick animal has nearly always been fatal, death being primarily due to its solvent action upon the red blood cells. In sick animals so treated there is a sudden rise of temperature, followed by an equally sudden fall, which is associated with collapse and death. On *post-mortem* the urine is found to be bloody, and the fat, subcutaneous tissues, and mucous membranes are intensely yellow from staining with altered blood pigment. These appearances follow the administration of serum not only in cases in which the disease has become well established and many parasites are present in the blood, but even when it is given before any parasites appear in the circulation, and while the only indication of approaching indisposition is an elevation of temperature. To discuss the scientific bearing of these phenomena would occupy too much space and would be somewhat beyond the scope of this report. I will therefore defer such discussion until the final report is prepared.

The employment of serum for therapeutic purposes having been found to be highly dangerous if large doses were given, the administration of repeated small doses was tried. By this means in some cases we succeeded in banishing the parasites without producing either hæmolysis or yellowness of the

mucous membranes ; nevertheless the animals died. *Post-mortem examination* in these cases showed that the pathological changes which had taken place in the kidneys, liver, and lymphatic glands were of such a character that recovery was out of the question. Only when animals were treated with small doses of serum in the initial stages of the disease were we occasionally able to save a few, but in practice such a method would be inapplicable, as treatment could only be begun in time if systematic temperature records were kept of every animal, and microscopic examination of their blood made from time to time.

Preventive treatment was also attempted by means of serum inoculation. In some cases a single large dose was injected while the animals were healthy, and in others repeated small doses were given, the animals subsequently being turned into the infected veldt in the hope that natural infection would produce, in any animal protected by serum injection, an attack of such moderate intensity that recovery and subsequent immunity would follow. Three animals treated with large single doses of serum and afterwards turned out all sickened and died, although the prolonged duration of their illness showed that in their case the serum exercised a certain inhibitive action. In other animals subjected to repeated doses no beneficial result followed the administration of doses of 5 cc., but repeated injections of 10 and 20 cc. had marked but varied results, much, apparently, depending upon the particular sample of serum employed. In one experiment, for instance, with serum taken from one particular animal out of eleven animals inoculated, six ultimately became immune, while with another sample of serum we were only able to save one animal in ten, and 50 cc. of another sample of moderately powerful serum saved and immunised three animals out of six.

It is possible that by preparing a more powerful serum, which would have taken a longer time, and by other modifications of the process, better results might have been obtained, but in the course of our work we found we had to reckon with a complicating factor which interfered most materially with any satisfactory application of serum treatment ; this factor is the occurrence of Texas fever complications. Texas fever or redwater infection, I have found in the course of our experimental work here, is much more widely disseminated in Rhodesia than I at first suspected, in fact, to such an extent does it exist that we need not be at all surprised if any Rhodesian animal develops an acute attack of Texas fever or redwater when attacked by any febrile disorder of a debilitating nature.

In former reports I have referred to cases in which coast fever has been complicated by redwater, induced as a result of the lowered vitality and elevated temperature caused by the first-mentioned disease. The same complications were encountered in the course of our serum experiments. In several cases in which animals attacked by coast fever appeared to be approaching convalescence after serum treatment, their temperatures having subsided and the parasites of coast fever having almost entirely disappeared, a sudden rise of temperature occurred which heralded the appearance of the organisms of redwater in the blood, the urine became bloody, and the animal, already weakened by the attack of coast fever, collapsed and died.

In this manner we lost no fewer than fourteen animals which presented every appearance of rallying from attacks of coast fever, mitigated as these were by the administration of serum, and there is no doubt that, in the absence of Texas fever infection, serum treatment would have been much more satisfactory.

In our second serum experiment in the course of which only one animal survived out of ten, death in several instances was due to Texas fever complications.

This experience shows that any system of preventive inoculation against coast fever is likely to be attended by a high mortality if it is based upon the production of a severe attack of the disease which is to be artificially controlled by serum treatment, or in any other way, as such severe attacks are likely to be followed only too frequently by fatal attacks of redwater. The best way to avoid this grave danger is to have recourse to some method of inoculation which will only produce such modified attacks of the disease as are not likely to have an unduly lowering effect upon the constitution of the animals subjected to it, and mild attacks of this description are produced by repeated blood inoculations. In no instance where blood inoculation was tried did we lose an animal by reason of the appearance of Texas fever, and, therefore, I am convinced that this method is at present the only practical one which can be profitably employed for the protection of susceptible animals.

Our third group of experiments were made upon animals believed to have become immune under natural conditions.

Of these experiments I may instance the following :—

(a) Two half-bred Zebu cattle were presented to the Station by Dr Sauer, who thought that cattle of this class might be found to possess a natural immunity to coast fever. Both animals were turned out to graze upon our infected veldt, and shortly afterwards they became sick and died.

(b) Five animals bought in Biera were brought to Bulawayo and run upon infected veldt. Of these, three sickened and died, two being young animals of pure Zebu breed. The survivors, old cows, have remained perfectly healthy, and have given birth to strong and vigorous calves, and the blood of one of these cows has been used for immunisation purposes with good results.

(c) Six animals presented by the German Government were sent to Bulawayo from Dar-es-Salaam for test. None have contracted African coast fever, but two have died from other causes, one being infected with tsetse.

(d) A number of animals (some, the survivors of various Salisbury herds; some, animals which had been subjected to inoculation at Hillside Camp with blood taken from Texan cattle previous to my arrival, all of whom had been grazing for many months upon infected veldt in Salisbury and Bulawayo) were turned out on our artificially infected pasture. None of these have sickened.

From these observations we may conclude :—

1st.—Zebu cattle have no inherent immunity.

2nd.—German East Africa cattle from coast districts are perfectly immune, and also some Biera animals, a fact bearing out my surmise that this disease is of coastal origin.

3rd.—The resistance displayed by naturally immunised animals indicates that such animals may be depended upon to withstand infection. Up to the present time we have not met with a single case of relapse in recovered animals, and we may, therefore, assume that recurrence of the disease only occurs in rare instances.

I have now studied this disease in all directions, so far as this was possible, for six months, and have made numerous and exhaustive experiments, therefore I believe I am sufficiently well informed to be in a position to express an opinion as to what means are best adapted for combating it.

In this relation, naturally the question of the feasibility of stamping out the disease presents itself. That this might be possible under certain conditions I believe, as with the assistance of the microscope we are able to identify all animals capable of spreading infection, not only animals which are sick, but also animals which have recovered from the disease and are still able to infect indirectly. Animals of the latter class, which may retain

the specific organism of the disease in their circulation, perhaps for the remainder of their lives, may be regarded as the real carriers of infection. How these animals may be disposed of demands consideration if the eradication of the disease is contemplated. With such an end in view it would be necessary to place recovered animals in such a position that they would no longer be a danger to others. This would not be so costly a process as it would be in dealing with some other diseases, as there would be no necessity to destroy such animals summarily. It would suffice if they were separated from their susceptible neighbours, and subsequently used for slaughter purposes. The position, therefore, somewhat resembles the position with regard to glanders, in which disease certain infected animals are identified by means of the mallein test, and are subsequently destroyed. Combating the spread of coast fever is in some respects less difficult than combating the spread of glanders, as the necessary measures would be simple, more certain, and less expensive.

But there is one condition which must be fulfilled if such a measure is to succeed, and that condition entails absolute control of all cattle and of all movements of stock.

Under European conditions, where this is feasible, stamping out a disease of this class would be the best way of dealing with it, although such a process would be expensive and would take some years to carry out. Here, however, in Rhodesia, conditions differ altogether from those obtained in European countries, as native cattle cannot be placed under control. Chief Veterinary Surgeon Gray, who is well acquainted with local conditions, assures me that such a stamping out process would be altogether impracticable in the present unfenced condition of the country, and impossible of application to native cattle, therefore another way out of our present difficulties must be sought, and the only one available is through artificial immunisation. Immunisation of cattle against African coast fever, it should be remembered, differs in one very important respect from immunisation against rinderpest and lung-sickness. Animals inoculated against lung-sickness and rinderpest do not become a future source of danger to susceptible cattle in their vicinity, but in all malarial diseases of the class to which African coast fever, Texas fever, and tsetse disease belong, immunised animals, although not directly capable of communicating infection, are indirectly able to do it with the assistance of an intermediate insect bearer, which, in the case of African coast fever, is the tick. Such immunised animals, while they remain in apparent health, are still able to disseminate infection, and that this is the case seriously handicaps those interested in stock raising, for it means that their surplus animals are not available for export alive, nor can pedigree animals be introduced from without unless they are subjected to an immunising process, and the most practicable process of immunisation against African coast fever which I can recommend at present is that of inoculation with recovered blood.

Were it not for the fact that the country is threatened by an epidemic invasion of African coast fever, and that the rainy season is approaching, when a recrudescence of the disease may be expected, I would have preferred to devote more time to the investigation of the merits and demerits of this method before expressing an opinion about it, but under present conditions I consider that circumstances justify my bringing the matter forward without delay, although it has not yet been gone fully into.

Every method of immunisation requires time before its benefits are apparent, and the establishment of immunity in African coast fever will take, I think, from four to six weeks. To wait, therefore, for the opening of the rainy season, when the spread of infection will be much more rapid, before commencing operations, would probably mean that many more animals would be

lost, as the inoculation of infected animals will be without beneficial effect. The process recommended is devoid of risk and is inexpensive. For the present I advise that it be only applied to animals exposed to imminent risk of infection, to infected herds and to animals running on infected pastures, particularly to herds in which isolated cases of the disease have only recently occurred. The work of carrying out such inoculations should, when possible, be left to veterinary surgeons or to those who have been instructed in the method. The animals used for taking the blood intended for inoculation purposes should be in apparent good health and condition, and should be either animals which have recovered from an unmistakable attack of the disease, or animals which have survived an outbreak which has carried off the majority of their neighbours, and which have subsequently grazed for a long time upon veldt known to be highly infected, and, when possible, the blood should be first microscopically examined before use, in order to determine whether the organisms usually found in the blood of recovered animals are present.

For the convenience of farmers and stockowners who are so situated that they cannot obtain the assistance of a veterinary surgeon, and who are unable to get an object lesson in the process of inoculating, the following directions are appended:—

To Inoculate.—The operator should provide himself with the following appliances:—

A lancet or sharp-pointed knife.

A trocar with canula about one-eighth of an inch in diameter, or a canula of this size pointed in the same manner as a hypodermic needle, to draw off the blood which is to be used for inoculating.

A wire egg whisk which has previously been cleaned by boiling or heating to redness in a fire.

A hypodermic syringe to contain 10 cc., which should have previously been boiled along with the needles, to be used for inoculating.

Two enamel pitchers to hold about half a gallon each, which have been rinsed first with a 5 per cent. solution of carbolic acid in water, and then washed out with boiled water.

A few pieces of freshly-boiled clean muslin to strain the blood.

To prepare it for the operation of bleeding, it should be cast, its legs secured, and the head held down. The jugular vein is then raised by passing a strong cord round the base of the neck; the hair should then be clipped off over the vein and the skin washed with soap, using a 5 per cent. solution of Jeyes' fluid or carbolic acid in water; then make a small incision over the vein along its length, with the knife cutting through the skin, insert the trocar and canula into the vein, passing the trocar upwards in the direction of the head, withdraw the trocar leaving the canula in the vein, and draw off the blood required into one of the enamel pitchers, stirring it all the time with the wire whisk to prevent it from coagulating.

When a sufficient quantity of blood has been taken, the cord should be loosened from the neck and the canula withdrawn. The wound may then be closed by placing a piece of adhesive plaster over it, by putting a stitch through the skin on either side with a suture needle and suture silk, or by pinning the lips of the wound together with a pin, which should only pass through the skin, and then twisting a piece of silk around the pin figure-of-eight fashion.

After stirring the blood for ten or twelve minutes it should be strained through a piece of clean muslin into the other enamel pitcher, and carefully covered to keep out dust and flies; it will then be fit for use.

As a pint and three-quarters of strained blood is sufficient to inoculate one hundred head of cattle, it will seldom be necessary to draw more blood from

an animal at one time than three pints, which, when whisked up and strained, will give at least 1000 cc. suitable for inoculating with, although, if necessary, three or four quarts can be taken from an animal without injury. Every care should be taken to cleanse the site of the operation before bleeding, and the vein should be injured as little as possible when the trocar is inserted.

In cases of urgency, where a trocar and canula cannot be procured, the jugular vein may be raised in the ordinary way and the animal bled with a fleam, but by so doing there is more risk of damaging the vein than if a canula is used, and the blood is more likely to be contaminated.

To inoculate animals which it is desired to protect, the animal is secured, the syringe is filled with strained blood, the loose skin of the neck is gathered up between the thumb and forefinger, the needle of the hypodermic syringe is inserted under the skin, 10 cc. are injected, the needle is withdrawn, the swelling caused by the injected blood is rubbed gently away with the hand, and the animal is released. The dose for all animals is 10 cc., irrespective of age.

In conclusion, I wish to impress upon those intending to inoculate, that at this stage of our work, when the whole question has not been gone thoroughly into, too much must not be expected of the method, nor must good results be looked for when it is applied to herds in which the disease has gained a thorough foothold.



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